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CEREBRAL SUBDURAL HEMATOMAS*

A Study of Three Hundred and Ten Verified Cases

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IN 1933, I[1] read a paper before this society on the diagnosis and treatment of subdural hematomas. This was based on my experience with 62 cases. By 1938, the number of cases that I had studied had increased to 215.[2] By January, 1941, 95 more cases had brought the total to 310. It is this group that forms the subject of this paper. As my experience has increased, it has become more and more apparent that the accuracy of one's conclusions varies directly with the number of cases from which the conclusions have been drawn. This trite remark is justified by the persistence with which small numbers of cases are published and made to serve as bases for conclusions that are in disagreement with or ignore those drawn from larger and more adequate series.[2–4] Notable among the exceptions to this statement are papers by Browder and his co-workers,[5] Davidoff and Dyke[6] and Ingraham and Heyl.[7] Examples of its truth are a recent editorial[8] based on two articles in one of which the author's experience was limited to 48 patients, the other being a synthetic report of 42 cases of which the author himself had seen 2, and a case report[9] presumably published to help care for injured naval personnel. The last is said to describe a case of "pachymeningitis hemorrhagica," but it presents all the autopsy findings of an intracortical clot. There is no doubt in my mind that personal experience with no less than 100 cases of significant subdural hematomas is indispensable to a competent opinion about the incidence, classification, pathology, diagnosis, treatment and prognosis of this very common condition.

In all the 310 cases reported herewith, the presence of the clot was verified either at operation or at autopsy, and was the only significant, or in multiple lesions, the most significant, intracranial lesion present. Although the clots were not weighed and the amount of subdural fluid was only roughly estimated, the size of the former and the amount of the latter were always pathologically significant. No case has been included in which the clot or the fluid could, by any stretch of the imagination, be considered incidental either to other findings or in their own right.

The gross pathology of subdural hematomas has been described by Leary and Edwards,[10] Leary[11] and Peet and Kahn.[12] Their work, especially that done by Leary, supersedes, amplifies and corrects the findings published by Putnam and Cushing.[13] Gardner[14] has covered the experimental field; those unfamiliar with his work should consult the original article. It is fundamental and unquestionably accurate. The surgical pathology has been covered by Munro and Merritt[15]; increasing experience has confirmed these earlier findings. So far as I know, no comparable studies have been published since then. I have no doubt, therefore, from these data and other evidence, as well as from my own increasing experience, that cerebral and cerebellar subdural hematomas originate as blood and cerebrospinal fluid mixed in varying amounts and incarcerated within the intracranial subdural space.

In conformity with what appears to be a fixed determination on the part of the medical profession to divide subdural hematomas into acute and chronic groups, the cases in this series have been grouped accordingly. However, since attempts to measure the acuteness by elapsed time are as unsatisfactory as they are inaccurate, in both the acute and chronic cases, I have classed those cases as "chronic" in which there was *no* evidence of fresh or unhealed associated brain injury. Cases that still showed evidence of unhealed or acute brain injury have been classed as "acute subdural hematomas."

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Since all hematomas are acute at the start and since they become chronic only because their presence has been unrecognized earlier by the attending surgeon, this distinction, although perhaps valid, seems to me to be unnecessary and artificial.

Chronic Solid Subdural Hematomas

If the hematoma is formed of pure blood, such as that resulting from the rupture of a bridging vein, and if there is no tear in the arachnoid, the hematoma will be expansile only while the bleeding is unchecked. After the bleeding has stopped, the hematoma will clot, and the clot will be encapsulated between two membranes by the growth of fibroblasts on both the dural and arachnoidal surfaces, after which organization of the clot itself takes place from the two membranes. Depending on the relative speeds of organization of the surface and liquefaction of the center of the clot, the latter will be replaced by, at one extreme, a single fibrous sheet representing the fused membranes and organized intervening clot and, at the other, by a collection of fluid with a very high protein content contained entirely within membranous walls that are fibrous and may include calcium. The former is adherent to the dura, and even on microscopic examination may be indistinguishable from it except as a local thickening. The latter appears as a cyst with numerous vascular and fibrous adhesions between its wall and the dura, arachnoid and pia.[10,11,15] It lies completely within the subdural space. Neither of these lesions is expanding. When they cause symptoms of increased intracranial pressure, they do so only because the other contents of the skull have been unable to accommodate themselves to this extra space-occupying lesion. This is the typical subdural hematoma associated with intracranial injury of the newborn[2] and is the type that develops from nontraumatic cases, — for example, in scurvy and blood dyscrasias, — as well as after craniocerebral injury. It is not necessarily an immediately fatal lesion even when associated with trauma. The patient is apt to have no significant symptoms at the time it is first formed, the injury is liable to be trivial, and the symptoms, when they do develop, are prone to be remissive. Until recently, clots that originated in trauma have usually been overlooked at the start and then recognized later only in the course of an investigation of the patient as a "brain-tumor suspect." This has been well emphasized by Horrax and Poppen.[16] It is this group of subdural hematomas that, after having been overlooked and undiagnosed at their formation, have been discovered later and described as "chronic subdural hematomas." If the diagnosis has been even more inaccurate, the patients have been committed to insane hospitals as psychotic, only to have the true cause of their mental abnormalities first recognized at autopsy.[17] This kind of clot was the subject of Putnam and Cushing's[13] paper, and is the least common of all intracranial subdural hematomas.

Chronic solid subdural hematomas occurred forty-five times in this group of 310 verified cases (Table 1). In 3 cases, an associated diagnosis of "old contusion of the brain" could be made. However, in these, as well as in the other 42 cases, any associated

Table 1. *Cerebral Intracranial and Cranial Complications.*

Type of Lesion	Cranial Injury			Other Hemorrhage			Acute Cerebral Injury				Chronic Cerebral Injury					
	Simple Fracture	Depressed Fracture	Compound Fracture	Extradural	Subcortical	Ventricular	None	Edema	Contusion or Laceration	Hyperthermia	Convulsive Seizures	Adhesive Arachnoiditis	Encephalopathy	Optic Neuritis	Hysteroneurosis	Toxic Dehydration
Acute mixed subdural hematomas (194 cases)																
Living patients	23	7	3	7	3	1	10	5	100	0	4	1	11			5
Dead patients	22	0	0	4	4	0	0	2	77	1	0	1	2			1
Totals	45	7	3	11	7	1	10	7	177	1	4	2	13	0	0	6
Chronic subdural hematomas																
Fluid type (71 cases)																
Living patients	2						67				14	3	9	1	3	
Dead patients	0						4				0	0	1	0	0	
Totals	2	0	0	0	0	0	71	0	0	0	14	3	10	1	3	0
Solid type (45 cases)																
Living patients	3						27				2	1	3	1		
Dead patients	0						15				3	0	4	0		
Totals	3	0	0	0	0	0	42	0	0	0	5	1	7	1	0	0
Both types (116 cases)																
Living patients	5						94				16	4	12	2	3	
Dead patients	0						19				3	0	5	0	0	
Totals	5	0	0	0	0	0	113	0	0	0	19	4	17	2	3	0

brain injury that had been present when the clot formed had long since healed by the time the hematoma had been diagnosed. Three cases had demonstrable fractures of the skull. Thirty-eight clots were solid and encapsulated, and seven were only a trivial craniocerebral injury — many being unaccompanied by unconsciousness even — and the subsequent persistent but often intermittent headaches. Objective symptoms are minimal and, if present, are usually caused by the associated brain

TABLE 2. *Cause of Death.*

TYPE OF LESION	CEREBRAL CONDITIONS		CIRCULATORY CONDITIONS			INFECTIONS					SURGICAL SHOCK	UNKNOWN
	MEDULLARY EDEMA	HYPERTHERMIA	CIRCULATORY FAILURE	ARTERIOSCLEROSIS	VASCULAR HYPERTENSION	PNEUMONIA	MENINGITIS	SEPTIC WOUND	SUBDURAL ABSCESS	EMPYEMA		
Acute mixed subdural hematomas	33	1	25	12	4	7	5	4	0	1	4	1
Chronic subdural hematomas												
Fluid type	0		2	0	0	2		0	0			
Solid type	4		8	3	1	2		2	1			
Both types	4	0	10	3	1	4	0	2	1	0	0	0

cystic and encapsulated, as described above. In 1 case, two solid clots were present. They were discrete, superimposed one on the other and both in the right parietal region. A fresh hematoma was present in addition to a chronic one — both being solid clots — in 1 case. In 1 case, the clot was calcified, and there was 1 in which simultaneous damage and toxic dehydration. Later on, the subjective symptoms and signs are those of a brain-tumor suspect. There is not necessarily any evidence of increased intracranial pressure. The diagnostic use of air in these so-called "chronic" cases by both the ventricular and lumbar subarachnoid routes has been recommended by Kun-

TABLE 3. *Location, Type and Identifiable Source of Clot.*

TYPE OF LESION	LOCATION OF CLOT					TYPE OF CLOT						IDENTIFIABLE SOURCE OF BLEEDING	
	UNILATERAL	BILATERAL	CEREBRAL	CEREBELLAR	NOT GIVEN	RECURRENT	OLD AND NEW	MULTIPLE	SOLID	CYSTIC	FLUID	CALCIFIED	RUPTURED BRIDGING VEIN	RUPTURED VENOUS SINUS
Acute mixed subdural hematomas														
Living patients	75	19	115	0	0	4	5				21			0
Dead patients	58	14	76	1	2	4	2				4			2
Totals	133	33	191	1	2	8	7	0	0	0	25	0	0	2
Chronic subdural hematomas														
Fluid type														
Living patients			67								67			
Dead patients			4								4			
Totals	0	0	71	0	0	0	0	0	0	0	71	0	0	0
Solid type														
Living patients	13	7	30			1	1	1	26	4		0	0	
Dead patients	10	5	15			2	0	0	12	3		1	2	
Totals	23	12	45	0	0	3	1	1	38	7	0	1	2	0
Both types														
Living patients	13	7	97			1	1	1	26	4	67	0	0	
Dead patients	10	5	19			2	0	0	12	3	4	1	2	
Totals	23	12	116	0	0	3	1	1	38	7	71	1	2	0

right parietal, right occipital and right frontal clots were present, the parietal clot recurring after removal. A ruptured bridging vein was identified as the certain source of the clot in 2 cases. Both these patients died of circulatory failure and were autopsied, one having been operated on previously.

Diagnosis

Early diagnosis of this type of hematoma depends on the recognition of a significant cause-and-effect relation between what may be considered

kel and Dandy,[18] Browder et al.,[5] Horrax and Poppen[16] and, doubtless, many others. It is unquestionably of great and often indispensable aid. I have never approved either the need or the safety of its use in the acute phase, however, preferring bilateral transtemporal exploration.

Treatment

Treatment is surgical, with removal of the solid, partially organized clot, and as much as is safe of the fused membrane that is the end result of com-

plete organization. Cysts should be emptied, and as much of the walls removed as is compatible with safety. In children, it is essential, as Ingraham and Heyl[7] have shown, that the membrane, and especially the one that lies on the arachnoidal side of the clot, be fully removed if later stunting of the growth of the hemispheres is to be prevented. The operation, which should be done by way of a flap craniotomy, is apt to be bloody, and postoperative clots and severe cerebral edema are frequent complications.

Immediate Results

There were 15 deaths in this group of 45 cases — a mortality of 33 per cent. All except 1 patient were operated on. The causes of death are interesting (Table 2). Circulatory failure was the commonest. This was present nine times. Medullary edema was considered to be the cause of death in 3 cases, hydrocephalus in 1, a recurrent clot in 1, and lobar pneumonia in 1. The clots were present on the left, the right and bilaterally each five times. Two of the hematomas recurred after operation, and another was calcified. Twelve deaths occurred in the group of solid clots, and 3 in the cystic-clot group (Table 3). Two of the latter were the result of birth injuries, in one of which, no operation was performed, and the other was present in a man of sixty-seven. In this connection, it may be noted that the average age of this group of fatal cases was fifty years, the youngest being ten months and the oldest sixty-seven years (Table 4).

TABLE 4. *Age Groups.*

TYPE OF LESION	YOUNGEST	OLDEST	AVERAGE	NUMBER OF PATIENTS
Acute mixed subdural hematomas				
Living patients	4 months	68 years	40 years	115
Dead patients	11 months	77 years	50 years	79
All patients			44 years	194
Chronic subdural hematomas				
Fluid type				
Living patients	5 months	62 years	30 years	67
Dead patients	8½ years	5 years	31 years	4
All patients			30 years	71
Solid type				
Living patients	9 weeks	66 years	38 years	30
Dead patients	10 months	67 years	50 years	15
All patients			42 years	45
Both types				
Living patients	9 weeks	66 years	38 years	97
Dead patients	10 months	67 years	40.5 years	19
All patients			40.5 years	116

All the 30 living patients had been operated on. Ten clots were on the left side, thirteen on the right, and seven bilateral. One recurred and was removed at the second operation (Table 3). Although the youngest patient was nine weeks and the oldest sixty-six years of age, the average age of this group was twelve years less than that of the fatal cases — thirty-eight as compared with fifty years. It seems fair to conclude that the prognosis in the group of chronic solid cerebral subdural hematomas is altered to some extent by the age of the patient but not by any other specific factor. It is impossible to determine the rate of incidence of this class of cerebral subdural hematomas among craniocerebral injuries in general.

ACUTE MIXED SUBDURAL HEMATOMAS

When both blood and cerebrospinal fluid collect and mix in the intracranial subdural space, the appearance of the resulting clot varies within wide limits. Macerated cortical tissue may also be added to the mixture. If the blood is at a maximum and the cerebrospinal fluid at a minimum, the clot closely resembles that described above as an "acute solid hematoma" — the variation being chiefly in the length of time that elapses before the blood solidifies. If, on the other hand, there is a maximum of cerebrospinal fluid, and a minimum of blood, the mixture and subsequent solution of the latter in the former preclude all attempts on the part of the dura to organize the hematoma. The hematoma is represented, therefore, by a collection of pigmented fluid with a high protein content. It is incarcerated in the subdural space but is, nevertheless, free to move anywhere within that space. It requires no great stretch of the imagination to visualize the change that variations in the relative concentration of blood and cerebrospinal fluid might make in color, consistence, and rate and completeness of coagulation in this type of hematoma. So long as coagulation and, therefore, organization by encapsulation do not take place, this blood dissolves in the cerebrospinal fluid that has escaped into the subdural space. This solution is separated by a dialyzing membrane — the arachnoid — from another solution (the cerebrospinal fluid) that has a relatively low protein content and a low osmotic pressure in relation to the hematoma. Under such circumstances, osmosis, with migration of the fluid of low protein content across the membrane to, and consequent dilution of, the solution of high protein content, is inevitable. With this movement and as the direct result of it, there is of necessity an increase in the amount of the high protein solution. This type of subdural hematoma is therefore an expanding lesion during the periods of solution and osmosis. The rate of expansion, which is rapid for the first five weeks, gradually decreases over the next two months as the concentration of protein in the two solutions approaches equality. This sequence of events has been demonstrated experimentally by Gardner[14] and by a graphic study of eighty-nine such fluids by Munro.[2] Browder et al.[5] consider my "theoretic explanation

that a subdural hematoma may be augmented by osmosis in a certain number of such lesions" entirely logical. They find it difficult, however, to explain certain variations in the consistence and color of different clots that have been present the same length of time after the injury. This finding has been my experience also, but unlike them I find no difficulty in accepting the explanation that, although the time interval is the same, the relative amounts of blood, cerebrospinal fluid and cortex are highly individual and should therefore produce hematomas no two of which can be alike.

This mixed type of subdural hematoma occurs exclusively as the result of craniocerebral injuries. In addition to damage to blood vessels on the brain surface, there is always a tear in the arachnoid. Thus, as noted above, in addition to blood, cerebrospinal fluid is discharged in varying amounts into the subdural space. The blood is commonly from cortical veins, less often from the large venous sinuses and less often still from cortical arteries. I have never seen the latter, but Browder and his associates[5] report its occurrence. If the injury has produced a laceration of the pia and the brain, cortical tissue may also be added to the blood and cerebrospinal fluid. Since these patients have, as a rule, severe brain injuries in addition to their hematomas, since many of them are in surgical shock for a varying period after the receipt of the injury and since these hematomas are expanding lesions, there is an associated compression of the brain that is beyond that which may be produced by uncomplicated cerebral injury. This group of patients has the highest fatality rate. In general, they are the ones who are first treated for a lacerated or contused brain but who despite that therapy get worse and often die with the clot unoperated on and undiagnosed. The prognosis is not invariably fatal, however, even in the absence of active therapy. Many patients, especially those whose hematomas contain less rather than more blood in relation to the amount of cerebrospinal fluid, recover from the immediate effects to become chronic invalids until such time as their fluid subdural hematomas or, as many are improperly called, their "hydromas" are removed. Early diagnosis and early treatment of the mixed subdural hematomas obviate this difficulty and save many lives in addition. It is this mixed type of subdural hematoma that is far and away the commonest of the subdural clots. If real progress is to be made in the reduction of the immediate mortality of craniocerebral injuries and the elimination of later disabling post-traumatic neuroses and invalidism, the attention of the medical profession must be concentrated on this group of cases to a much greater degree than it has been in the past.

In this group of 310 cases, 194 had acute mixed subdural hematomas. These were complicated by a recognized associated major brain injury in 184 cases. Its presence was verified at operation or autopsy in every case. No note of any brain injury was made in the other 10 cases. A fracture of the skull was demonstrated in 55. Forty-five of these were simple, seven were depressed, and three were compound fractures. Nineteen cases had other major complicating hemorrhages. Eleven were extradural, seven subcortical, and one ventricular. Toxic dehydration was present in 6 cases. No clots could be traced to ruptured bridging veins (Table 1). Six patients had infected wounds.

Diagnosis

I am convinced that a certain diagnosis of the presence of a mixed subdural hematoma can be made only by exploratory trephination. The next most accurate method is by either an encephalogram or a ventriculogram. I consider an encephalogram too dangerous a method to be used in these acute cases, and mention it only to condemn it. I see little to choose between the risk of a ventriculogram and an exploratory trephination. Since my experience leads me to believe that explorations made in areas other than the temporal miss a certain percentage of temporal and frontal clots and since exploration together with ventriculography forces the operator to make biparietal or bifrontal trephines, I have never felt justified in using air in this way in these cases. Aside from the operative demonstration of a subdural collection of blood, the most important diagnostic aid is to recognize the presence of a brain injury, treat it adequately, and then be prepared to carry out a transtemporal exploration if the patient fails to improve or gets worse under nonoperative therapy. Of course, before exploration, toxic dehydration must also be eliminated as a cause of the continued symptoms. The subjective and objective symptoms produced by these hematomas cannot be distinguished from those caused by either an uncomplicated brain injury or toxic dehydration. Lateralizing signs are notoriously inaccurate. The cerebrospinal fluid is altered from normal only because of the brain injury, and its pressure may be high, normal or low, even in the presence of a large clot. I have not seen any dependable regularity in the development of coma or a latent interval and do not regard their presence or absence as being of any diagnostic import. I have the impression that lasting mental changes and the development of a contralateral central facial palsy while the patient is under treatment are commoner in the presence of mixed subdural hematomas. Unilateral fixed dilatation of the pupil is a suggestive

diagnostic sign, but it is not reliable.[1] Exploratory trephination, transtemporal by preference and bilateral if necessary, is never contraindicated in a patient who has been knocked unconscious by a blow on the head and who is not getting better or is getting worse under adequate lumbar-puncture and fluid therapy.

Treatment

Just as in the cases of solid hematoma, the treatment of the acute mixed hematoma is exclusively surgical. The operation should not be done while the patient is in shock or toxic from dehydration, but otherwise should be carried out in accordance with the requirements noted above. When a hematoma is found and no matter what its physical consistence may be, it should be removed by suction as completely as possible after the majority of the squama and the adjoining edges of frontal and parietal bones have been removed and the dura opened to the limit of the bony windows. Afterward, the subdural space should be drained with rubber tissue and the dura left open. The rest of the wound should be closed in layers. Irrigation of the damaged cortex should be studiously avoided. The approach should be through a vertical muscle-splitting temporal incision. I consider bone flaps contraindicated and too dangerous. It should be noted, however, that Browder and his associates[5] advise and use small bone flaps with apparent satisfaction and success in this type of case. The postoperative care of these patients requires as much judgment and skill as the operative procedure — indeed, more in many cases. The brain injury must be treated, and in addition, the need for intelligent general supportive measures is more than ordinary. Finally, malignant postoperative edema is very prone to develop and unless treated with all the means at one's command will bring about a fatal ending to an otherwise successful case.

Immediate Results

Seventy-nine of the 194 patients with acute mixed subdural hematomas died in the hospital. This is a mortality of 41 per cent. Since all patients were operated on, the operative mortality is essentially the same. The causes of death in the fatal cases are interesting and particularly so when compared with the cause of death in the fatal cases of solid hematoma (Table 2). Circulatory failure was again the chief cause, having occurred in 29 cases. Next, came medullary edema, with 27. Pneumonia and meningitis caused 5 and 6 deaths respectively; in 4 of the 6 cases with meningeal infections, the complication occurred postoperatively. Surgical shock caused 4 deaths, recurrent unrecognized clots, 2, and respiratory obstruction, uremia and empyema thoracis, 1 each. In 1 case, the cause of death was not clear. Eleven patients died without operation. All were autopsied, and the cause of death determined thereby. Fifty-eight clots were unilateral, twenty-eight being on the right and thirty on the left. Fourteen were bilateral, four recurred, and 2 patients had a combination of old and new clots. There were no multiple clots. Four hematomas were fluid as distinct from the liquid and solid clots in that there was no gross solid material present. Seventy-six were cerebral, one was cerebellar, and the location of two was not given (Table 3). The youngest of the patients who died was eleven months, the oldest seventy-seven years, and the average age was fifty years. In 3 cases, the age was not known (Table 4). The immediate effects of the operative procedure, as represented by medullary edema, postoperative infections and circulatory incompetence, were major factors in determining the number of fatalities. Of these, the last is the only one with prognostic significance under the circumstances and, when taken in conjunction with the ten years' difference in the average age of the fatal and nonfatal groups, emphasizes again that the older the patient with a subdural hematoma, the worse the prognosis.

One hundred and fifteen patients with acute mixed subdural hematomas survived and left the hospital relieved of the symptoms that brought them in. In 75, the clot was unilateral, forty-one being on the right and thirty-four on the left. Nineteen were bilateral. In 5 cases, both old and new hematomas were present. In 1, they were bilateral — that is, four clots; in 1, one clot was on the right and the other on the left; in 1, both clots were on the right; in 1, both were on the left; and in 1, a fluid clot was present in association with a solid right-sided hematoma. Four clots recurred after operation — one bilaterally and three on the right side. Twenty-one were fluid in type, being unlimited in their freedom of movement within the cerebral subdural space. The total protein in 14 of these and in 2 of the 4 similar fatal cases ranged from 4020 to 56 mg. per 100 cc., with an average of 396 mg. In 3, the fluid clotted before it could be examined. All one hundred and fifteen hematomas were cerebral (Table 3). As in the group of solid hematomas, the average age — forty years — was ten years less than that of the corresponding patients who died. The youngest patient was four months, and the oldest sixty-eight years (Table 4).

Chronic Fluid Subdural Hematomas

When the fluid type of mixed subdural hematoma — that is, the type made up of a maximum

of cerebrospinal fluid and a minimum of blood—is allowed to go untreated until after the acute brain injury is healed, it may be said to have become chronic. This is in accordance with the definition of chronicity as given above. Depending on its size, the amount of dissolved blood, the percentage of protein originally contained within it and the length of time that has elapsed between its formation and its discovery or operative removal, it may still be either an expanding lesion and increasing in amount or fixed in relation to both its protein content and its size. In either event, it will be, as it has been from the beginning, incarcerated within the subdural space but free to move anywhere within those limits. I[19] have described these hematomas in detail elsewhere and need not repeat the description here. Others have also described them, but, in my opinion, incorrectly—as "hydromas." Hydroma or hygroma is defined as a "sac, cyst or bursa distended with a fluid."[20] This term is therefore only descriptive and carries no pathological significance. The term was first applied to subdural fluid accumulations by Naffziger[21] before the problem of subdural hematomas was understood. Its continued use, as advocated by da Costa and Adson[22] in the mistaken belief that a hydroma is a separate entity and not one form of subdural hematoma, is not justified. Their failure to explain how air put into the subarachnoid space can reach the subdural space and outline fluid therein *after* the tear in the arachnoid has closed and incarcerated the fluid, and particularly when the latter is enclosed within cyst walls as well, is a fatal omission. There seems to me to be a need for a clearer understanding of the entire subdural-hematoma problem rather than for the identification of another pathologic entity.

Suffice it to say that, just as with the chronic solid clots, so these chronic fluid clots would not exist had their presence been suspected and verified at the time of the original craniocerebral injury, because they are all the direct result of such injuries. This extremely disabling but nonfatal condition is always caused by an injury to the head. The symptoms are those commonly described as being caused by post-traumatic neurosis or malingering, and are usually associated with a severe and persistent true neurosis. If untreated, the patients become hypochondriacal invalids and permanent charges on the community. They do not go insane, nor do they die as the result of this condition.

This type of hematoma occurred in 71 of the group of 310 patients. None had any associated brain injury at the time of examination, although it was possible to demonstrate a simple fracture of the skull in 2 cases. Complications that were frequent in the other types of subdural hematomas were conspicuous by their absence. On the other hand, there was an unduly large preponderance of the type of condition that suggests permanent brain damage. For example, 14 cases had convulsive seizures, 10 had intellectual changes classified as "encephalopathy," 1 had optic neuritis, and 3 had hysteroneuroses (Table 1).

Diagnosis

A correct diagnosis can be made only at operation. Air injection is of no help, as has been shown by von Storch and Munro.[23] The cerebrospinal fluid is normal, as is the intracranial pressure. Objective and subjective signs and symptoms are conspicuous by their absence—their presence being significant from a prognostic rather than a diagnostic point of view, the occurrence of convulsive seizures being especially unfavorable. In general, the statements made in this regard in an earlier paper[19] still hold true.

Treatment

The treatment is surgical. The operation should be done as soon as possible after the receipt of the injury, since the shorter this interval the greater the chance for a permanent cure. The subdural space should be exposed through a transtemporal approach, as described above. The approach need be only on one side, preferably that opposite the speech cortex. As much as possible of the fluid hematoma should be removed, and the subdural space drained with rubber tissue. The wound is closed in layers except for the dura, which should be left open. There are usually no postoperative complications, and the patients have immediate and permanent relief so far as their associated neurosis and permanent cerebral changes allow.

Immediate Results

Four of these 71 patients died—2 of lobar pneumonia, 1 of progressive encephalomalacia and circulatory failure, and 1 of heart failure and chronic alcoholism (Table 2). Their ages were forty-three, fifty-nine, eight and a half and fifty-five years respectively—an average age of thirty-one years (Table 4).

Sixty-seven patients survived. Eight were not relieved of the symptoms for which they had been operated on. The youngest was five months, and the oldest sixty-two years, the average age being thirty years (Table 4). For what they are worth, the comparative figures demonstrate that in this type of hematoma, age has little effect on prognosis so far as mortality is concerned.

Discussion

This series of 310 major subdural hematomas demonstrates that no less than 62 per cent of intracranial subdural hematomas can be recognized

in their acute stage. If they are so recognized, the mortality can be expected to be in the neighborhood of 40 per cent.[5, 24] It varies with the patient's age and the efficiency of his circulation. So far as the individual types of hematomas go, the fatalities show a consistently higher age level. It seems reasonable to conclude that increasing age is a detriment to the patient in the acute phase of this disease but has little influence on the mortality when the chronic forms are considered. The lished.[2] The incidence in this particular group is 13 per cent, but my series is undoubtedly "loaded" because serious and operable cases gravitate toward and mild and nonoperative cases away from my service. I cannot account for the discrepancy between these figures and the 1 per cent incidence reported by Browder et al.[5]

My mortality, on the other hand, is essentially identical with his — 41 per cent. In my series, of course, this refers only to the acute mixed

TABLE 5. *Miscellaneous Complications.*

TYPE OF LESION	CIRCULATORY CONDITIONS			INFECTIONS					OTHER INJURIES			GENERAL CONDITIONS			MISCELLANEOUS CONDITIONS	
	ARTERIOSCLEROSIS	VASCULAR HYPERTENSION	HEART DISEASE	PNEUMONIA	EMPYEMA	SEPTIC WOUND	NEPHRITIS	OSTEOMYELITIS OF THE SPINE	FRACTURED PELVIS	FRACTURED SPINE	RUPTURED SPLEEN	ALCOHOLISM	SYPHILIS	AVITAMINOSIS	BRAIN-TUMOR SUSPECT	CHOREIORETINITIS
Acute mixed subdural hematomas																
Living patients	9	1	2	6	1	2	0	1	1	1	0	10	2	1		
Dead patients	12	4	0	7	1	4	2	0	0	0	1	6	2	0		
Totals	21	5	2	13	2	6	2	1	1	1	1	16	4	1	0	0
Chronic subdural hematomas																
Fluid type																
Living patients	1			0								1			1	
Dead patients	0			2								1			0	
Totals	1	0	0	2	0	0	0	0	0	0	0	2	0	0	1	0
Solid type																
Living patients	1	1	2	0		0						1	0		1	1
Dead patients	3	1	0	2		2						0	2		0	0
Totals	4	2	2	2	0	2	0	0	0	0	0	1	2	0	1	1
Both types																
Living patients	2	1	2	0		0						2	0		2	1
Dead patients	3	1	0	4		2						1	2		0	0
Totals	5	2	2	4	0	2	0	0	0	0	0	3	2	0	2	1

additional prognostic significance of inefficiency of the circulation is of equal importance. This is only too evident from the figures in Table 2 and particularly so if one adopts the broad point of view, which recognizes that medullary edema and hyperthermia are at least closely linked to changes in the circulation.

Miscellaneous complications occurring in this series are listed in Table 5. They are of no great significance except so far as alcoholism and syphilis are concerned. As is well known, both syphilis and alcoholism have been held to be major if not exclusive causes of cerebral subdural hematomas. This series demonstrates conclusively that their presence is merely coincidental. Their single possible etiologic connection has to do with alcoholism, and this is recognizable only because a person who is drunk is likelier to injure his head than a sober one.

Cerebral subdural hematomas may be expected to occur in about 10 per cent of all hospitalized craniocerebral injuries. This incidence is considerably less than the figures I have previously published.[2] The incidence in this particular group is 13 per cent, but my series is undoubtedly "loaded" because serious and operable cases gravitate toward and mild and nonoperative cases away from my service. I cannot account for the discrepancy between these figures and the 1 per cent incidence reported by Browder et al.[5]

My mortality, on the other hand, is essentially identical with his — 41 per cent. In my series, of course, this refers only to the acute mixed hematomas and predicates a willingness to do early and frequent diagnostic transtemporal trephinations.

No attempt has been made to determine the details of the causative accidents or the sex of the patient. An unrecognized and improperly treated subdural hematoma is as fatal in a woman as it is in a man, and it is of little moment, so far as diagnosis and prognosis are concerned, whether it followed an automobile accident, a kick by a horse or a fall from a window. The important thing is to treat the condition early and adequately, and thereby reduce to a minimum the so-called "chronic" hematomas with their associated disabling symptomatology and at the same time lower the mortality of the acute cases.

CONCLUSIONS

In general, it is undoubtedly true that the commonest intracranial subdural hematoma is the mixed type and that the cause for its formation is trauma to the head. The presence of a cerebral subdural hematoma, regardless of its variety, does

not necessarily lead to a fatality. When the patient survives the immediate effect of the injury, the hematomas inevitably cause later symptoms. These symptoms will be disabling, interfere with the economic efficiency of the patient, and lead to a permanent neurosis, which may be associated with permanent structural changes in the brain. Such tragic late effects are preventable if the physician who sees the patient at the time of the original craniocerebral trauma bears in mind the fact that if patients with only contusion and laceration of the brain are adequately and promptly treated they will get better and be freed of their major symptoms rapidly. If such patients fail to get better, or get worse, however, the responsibility rests with the physician to recognize the possibility that a mistake has been made in the diagnosis and that, as a result, he must investigate and treat the cause of this failure to recover by every means at his command.

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INSTRUCTION OF LAYMEN IN THE EMERGENCY SPLINTING OF FRACTURES

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ACCIDENTS do not happen at carefully chosen sites. They are apt to occur under conditions in which medical aid either cannot be obtained or can be had only after a prohibitive lapse of time. Such accidents take place in the woods or fields incidental to hunting and sports, or on highways in thinly settled districts. There is now the added possibility of large-scale public disaster, explosion, bombing or invasion in which medical facilities, although immediately available, are temporarily overwhelmed.

The present state of national emergency has stimulated in the general public an unprecedented interest in the handling of accidental injuries, an interest that is well attested by recent dramatizations of first-aid treatment in popular magazines. In the past, lay education along these lines has been neglected by the medical profession, with the exception of certain relatively small select groups, such as the Red Cross and Boy Scouts. Now comes a sudden demand on the profession for instruction of civilian-defense workers in the handling of emergency injuries. It is a challenge that must be met and is being met by doctors throughout the country, who are cheerfully giving their time and energy, chiefly under the auspices of the American Red Cross, to lecture and instruct lay groups in the emergency care of the injured.

To many a physician, the role of instructor will seem new and strange. To be really effective, his enthusiasm and willingness must be combined with thorough mastery of the subject matter and with some knowledge of the art of teaching. The physician, specialist as he is in the business of learning, trained to a keen observation and retentive memory, is prone to credit his hearers with similar aptitudes in assimilating and retaining facts presented to them. This is a common error that the instructor must ever bear in mind. He can avoid other pitfalls, and will add measurably to the effectiveness of his instruction by observance of the following general rules:

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Use simple language—burden your audience with as few technical terms as possible.

Repeat, and repeat again, a few essentials—do not try to cover a mass of details.

Emphasize basic principles so that the student will understand clearly what it is that he is trying to do.

In choosing among several methods of first-aid treatment, present only the simplest effective one.

Be practical—present actual in preference to hypothetical cases, and confine yourself to the real limitations of the case presented and be sure that the method described can be used in the situation under consideration.

Since fractures make up a great part of all accidental injuries, the problem of giving instruction in emergency splinting naturally assumes considerable importance. At first glance, the subject of splinting seems to be an easy one to teach. But as one examines it more closely, certain difficulties

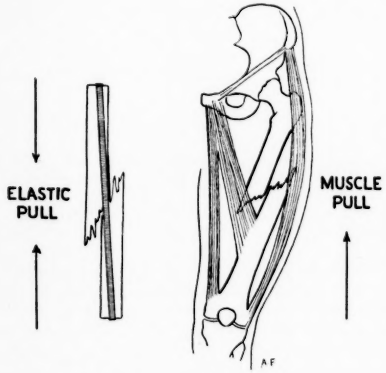

FIGURE 1. *The Pull of a Rubber Band over the Ends of a Broken Stick Illustrates Muscle Pull on a Broken Bone.*

appear. The rationale of splinting is, of course, simple to explain. The pull of a rubber band over the ends of a broken stick suggests the effect of muscle pull on a broken bone (Fig. 1). The rubber band causes each fragment of the stick to slip past the other; with a broken bone, on the other hand, the proximal end is fixed by the body so that the distal fragment tends to be drawn past the proximal. The potential damage to soft tissues by the knifelike ends of bone fragments that have not been splinted can be readily demonstrated, as can the salutary effect of a properly applied splint (Fig. 2). But, presumably, the layman will have no Thomas, Murray-Jones or Keller-Blake splint available, nor will he have at hand an adequate supply of flannel, muslin and gauze bandages, basswood splints, tongue blades, safety pins, rope and adhesive tape. What can he do?

It is no answer to urge that first-aid kits and splints be carried in every car and kept in every home. This worth-while objective may some day become a reality, but for the present it will not solve the first-aid worker's problem. Instead, he must be

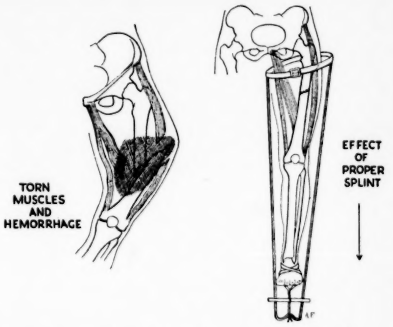

FIGURE 2. *Soft-Tissue Damage Resulting from Transportation without Splinting Compared with Bone Fragments Controlled by an Adequate Splint.*

shown that these materials, although desirable, are by no means essential and that adequate splinting can be achieved quite simply and effectively by using such things as may be at hand or close by. In short, he must be taught to improvise. This calls for ingenuity and imagination in the instructor quite as much as proficiency in technic and experience in teaching.

During the last six months, under the auspices of the New York and Brooklyn Regional Fracture Committee of the American College of Surgeons, a motion picture in color, demonstrating for the layman the emergency splinting of fractured arms, legs and spines, has been produced.* The film is designed to show how an effective splint can be improvised and applied by anyone in a few moments, under emergency conditions, only materials ordinarily available near the scene of an accident being used. The experience gained during the production of this picture may be helpful to others who have volunteered to teach first aid for fractures.

The methods of splinting described below are not necessarily the best, but they have been tested

*"Splint 'Em Where They Lie!" A 16-mm. Kodachrome motion picture in two parts. Part I: Fractures of the extremities (400 feet). Part II: Fractures of the spine (200 feet). The films may be obtained, through the author of this paper, for exhibition to lay or professional groups.

and found effective. Above all, they have one outstanding virtue: they are extremely simple, easily taught and well remembered. Modifications and improvements will undoubtedly occur to many. Certainly, no claim to originality is made. Rather, the basic idea throughout is to suggest what may be used for splinting and thus to stimulate the imagination and originality of others. The following description of emergency fracture care is an outline of the subject in a form suitable for presentation to lay groups.

Diagnosis

The first task the physician instructor has to face is teaching diagnosis. In most cases, the layman cannot be expected to know whether or not a fracture is present unless he makes some kind of examination. Besides, the victim may be unconscious or drunk, or too badly frightened, bewildered or hysterical to co-operate intelligently. More significant is the fact that an examination may disclose the presence of other fractures, which might easily be overlooked in the enthusiasm and excitement of getting to work on the victim. It is only natural for the novice to concentrate his attention on an obviously broken arm or leg, unaware that the victim has also sustained the far more serious injury of a fractured spine. The possible dire consequences of such an error can well be imagined. The layman must therefore be taught some simple but thorough method of examination, and the instructor should insist that he apply it before undertaking first-aid measures as such. Rarely do conditions justify a reversal of this procedure.

What should the examination include, and how comprehensive should it be? Specifically, just what should the layman be trained to look for? In fractures of the extremities, certain cardinal signs, such as local pain, tenderness, loss of function and gross deformity, are readily determined. Search for them can do the victim no harm, and their presence should at least make the layman suspect a fracture. He should also be taught that the peculiar grating sensation, known as crepitus, produced by broken bone fragments, and abnormal freedom of motion in one extremity compared with the opposite are major signs that are present in many fractures. But, at the same time, he must be strictly cautioned that many fractures exhibit neither the one nor the other and that attempts to elicit them are not only unnecessary but are to be severely condemned as being apt to do much harm.

Fractures of the spine often present special difficulties in diagnosis, even for the trained physician. Any great degree of accuracy in their recognition by laymen, therefore, is not to be expected. Local pain, tenderness and disability are inconstant and unreliable. Paralysis or anesthesia of the extremities, or both, if present, are significant but must by no means be considered essential for a diagnosis. After these facts have been pointed out to the layman, it is well to stress the value of treating as an actual spine fracture any injury to the neck or back that presents one or more of the signs mentioned above.

The layman must be taught the necessity of recognizing shock early and of becoming thoroughly familiar with its ordinary manifestations: pallor, weakness, faintness, cold clammy extremities, weak pulse and shallow respirations. He should approach the problem of diagnosis calmly and with assurance. He must take what time is necessary for a thorough and systematic, but gentle, examination. This is far more easily said than done at the scene of an actual accident. The volunteer is often subject to interference by anxious relatives and importunate bystanders, who demand action of some kind immediately and deprecate his efforts to ascertain first the nature and extent of the victim's injuries. The first-aid worker must not allow himself to become disconcerted by this well-meant but unfortunate pressure. He must proceed deliberately in spite of it until he has concluded a satisfactory examination and determined the number, kind and severity of the injuries present.

First-Aid Care

Fractures in General

The general condition of the victim of an accident should receive first attention. Following a major fracture, a person may or may not be in great pain; however, he is practically always suffering from some degree of shock, and simple measures to combat this condition should promptly be initiated. Among these may be mentioned covering the victim with coats or blankets to preserve body warmth. The administration of mild stimulants, such as hot coffee and tea, although advantageous, is usually impracticable under the circumstances of the accident. If the victim is kept warm from the outset, however, much will have been accomplished. In addition, splinting and immobilization of the fracture will, of themselves, tend still further to reduce shock. Active bleeding must, of course, be controlled at once, but this is a separate subject and lies outside the scope of the present discussion.

Regarding the emergency handling of compound fractures, physicians themselves are not agreed. My own conviction is that the layman should be taught the difference between a compound and a simple fracture and the reasons, chiefly potential contam-

ination and infection, for the greater seriousness of the former. He can also be shown the risk of converting a simple into a compound fracture by transporting the victim without proper splinting (Fig. 2). As a matter of practical expediency, I believe he should be taught to apply a traction splint to any fracture of a major extremity, simple or compound, before attempting to move the victim.

Fractures of Extremities

In a fracture of an arm or leg, while one person is concerning himself with the general condition of the victim, another should steady the fractured extremity. He should stay with the victim and continue the support while others seek a suitable splint. Material that will serve as a splint is usually the most difficult part of the required equipment to improvise under emergency conditions, and yet at times it may be surprisingly easy. A fence rail, signpost, fallen tree, broom, rake, rifle, skipole or ski is adequate, and other materials may suggest themselves. Under certain conditions, such as darkness, storm and freezing cold, more harm than good may result from the delay caused by seeking a splint, and it may be much wiser simply to bind one leg to the other or the arm to the body than to run the risk of increased delay and exposure to a seriously injured and weakened person. Each case must be considered on its merits, and if there is any real question between life and limb, the issue must, of course, be decided in favor of the former.

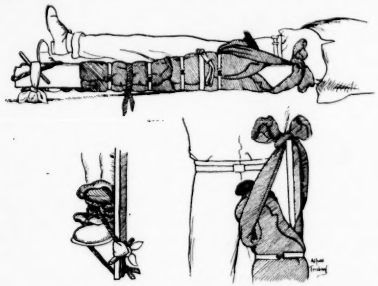

FIGURE 3. *Emergency Splint for Fractured Thigh or Leg, Improvised with an Old Board and Articles of Clothing.*

Ideally, the splint should be light but strong. It must be of sufficient length to reach from above the shoulder to well below the hand in a patient with a broken arm, or from above the hip to considerably below the foot in one with a fractured thigh or leg. It is much better for the splint to be too long than too short.

Application of the splint is a relatively simple matter. In place of bandages, rope and adhesive, various articles of clothing can be used effectively. For example, if an old board is used to splint a fractured thigh, it can be applied as follows (Fig. 3). A coat is slipped between the victim's thighs with the neck of the coat toward the crotch, and a loop is loosely fashioned about the upper portion of the hip by knotting the ends of the sleeves together. A folded hat is placed between coat and crotch to ease pressure against the latter when the splint is tightened. The upper end of the board is then engaged in the loop formed by the coatsleeves. At this point, a smooth, steady pull is exerted on the foot by the assistant, who has been steadying the injured extremity. This pull, once begun, must never be relaxed until the splint has been applied and finally adjusted.

Two neckties, knotted together to form one tie of double length, are looped over the ankle, which has been padded with another folded hat or other thick material. The neckties are then passed along each side of the foot and over the lower end of the splint, where they are tied tightly together. The loop thus formed is tightened by means of a small stick passed through it and twisted over and over. The stick is kept from untwisting by a handkerchief tied around it and the splint. The necktie loop should be tightened in this manner until the broken bone is steadied. Further tightening beyond this point is not only useless but dangerous to the circulation. Thus, traction, or pull, has been provided to overcome muscle spasm, but this is not enough.

The thigh and leg must be wrapped securely to the splint to support the extremity and prevent motion of the broken bone. The rest of the coat, whose sleeves are serving as a traction loop, is wrapped snugly about the thigh and splint. Garters, suspenders or neckties are passed around it and fastened for additional support. In similar fashion, the leg is wrapped to the splint, a vest, shirt or another coat being used. The splinted leg may then be lowered, but one must keep the heel of the foot off the ground by resting the lower end of the splint on a stone or other support. This avoids painful and dangerous motion of the bone fragments. It should be emphasized that only then is the fracture victim ready to be moved from where he has fallen. When the victim is picked up, the foot of the injured extremity should never be lifted; one should always lift the splint by which it is supported. Similarly, when the victim is placed in a car for transportation, a support for the end of the splint is arranged so that the foot hangs free, suspended by the splint. These simple precautions greatly minimize pain and shock.

A broken arm can be provided with an emergency splint in much the same way (Fig. 4). One assistant steadies the arm by taking a firm grasp of the hand. Pull on the arm must not be so strong as pull on the leg, since the muscles are weaker. A coat is slipped between the arm and the body, and its sleeves are looped above the shoulder, as described above. A folded hat or other pad is placed in the armpit to relieve pressure, and the upper end of the improvised splint is engaged in the coatsleeve loop. Neckties, looped over the wrist, which has been padded with another folded hat, are tied over the lower end of the splint and tightened with a small stick just sufficiently to steady the bones.* The stick is again held in place with a handkerchief tie, and another handkerchief, loosely tied about the splint, gives the hand support. The arm and forearm are then wrapped to the splint exactly as in the treatment of a broken thigh.

FIGURE 4. *Broken Arm in Emergency Broom Splint. Note that a coat is thrown over the body to keep the victim warm.*

Fractures of the Spine

When one presents this part of the subject, it is well to make sure that the layman understands clearly what the spine is, in general how it is constructed, and what purposes it serves. He should be shown, with a rough sketch or two, that it is made up of a number of small blocks of bone, of more or less equal size and shape, placed one on top of another in a tall column (Fig. 5). The instructor should point out that the function of the spine is not only to support the body but also to protect a large bundle of vital nerves — the spinal cord — that connect the brain with the rest of the body. This should be sufficient anatomy. Any attempt to give it in more detail will confuse the listener and interfere with a clear grasp of the subject. After all, time, as well as the average mental capacity, is limited.

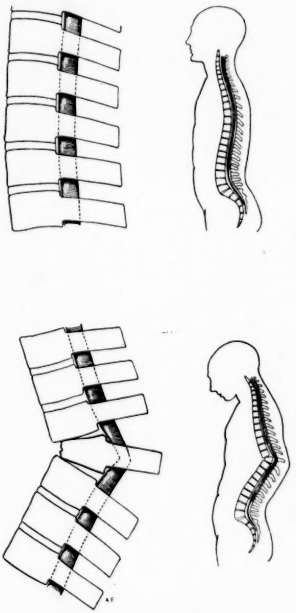

FIGURE 5. *Anatomy of the Spine and Cord, Simplified to Show the Mechanics of a Fracture of the Spine.*

Having laid this groundwork, one should explain the mechanics of injury. Sudden, violent for-

*It must be emphasized in no uncertain terms to a lay group that major blood vessels and nerves lie close to the surface in the armpit, where they are extremely vulnerable to pressure. Not only must the armpit be well protected with padding, but also great care must be taken not to apply excessive traction by turning the small stick up too tightly. Paralysis resulting from overpull is a real danger that must not be forgotten. Pull should not exceed 4 or 5 pounds, and when there is doubt, it is far better to have the Spanish windlass too loose than too tight.

ward bending of the spine, as in a fall from a height or in an automobile collision, should be stressed as the usual cause of such fractures. The possibility of damage to the cord should be given as sufficient reason for treating any suspected back injury as a fractured spine, and forward bending of the spine, during handling of the victim, should be avoided for the same reason.

Concerning the exact method the layman should be taught in dealing with a suspected spinal injury, there is room for some difference of opinion. Although details may vary, the underlying principles remain the same. Here again, in my opinion, simplicity should govern. After the victim's general condition has received attention, some strong flat support on which to place him must be found. This may be a board, shutter, door or other large flat object. It should be nearly body length or longer. Securing an adequate support of this kind often proves difficult, and it may be necessary to go some distance to find anything that will serve. Weather and the victim's general condition permitting, however, the effort should be made. Transportation of these injuries without proper support is extremely hazardous.

FIGURE 6. *Method of Moving a Victim with a Fracture of the Neck to an Improvised Stretcher.*

When the neck is injured, one person kneels facing the top of the victim's head and, grasping the sides of the face and jaw between his hands, steadies the head in line with the body (Fig. 6). No pull should be made on the head, and care must be taken not to bend the neck forward. An assistant supports the legs by holding the feet. A third helper then reaches across the door or other support that has been placed beside the victim and takes a firm grasp of the clothing at hip and shoulder. At a given signal, the victim is slid sidewise onto the door. All assistants must work smoothly together so as not to twist or bend the victim's neck. Support of the head must not be relinquished once the victim has been placed on the improvised stretcher but must be maintained throughout transportation. The door, with the injured person on it, may then be lifted and placed in a truck or wagon for conveyance to a hospital.

When the back is injured and the victim lies face down, he may simply be pulled sidewise onto the door by two people working together, one grasping the feet and the other the clothes at hip and shoulder, as described above. If the victim happens to lie face up, his arms should be brought above his head, and he should be rolled slowly over on the door, which has been placed on the ground alongside him. When this is done, it is essential to keep the trunk and extremities in line so that they are moved as one piece. In this way, the victim's back is not twisted or bent, and danger to the spinal cord is avoided.

When a rigid support such as a door cannot be found, an automobile robe or blanket, if available, may be used for conveyance of the victim, as follows. If a neck fracture is suspected and the victim lies face up, the blanket, rolled tightly lengthwise for about two thirds its width, is placed alongside him with the rolled portion next to his body. One assistant steadies the head in line with the body, as before, while one or two others tuck the blanket roll carefully under the victim and gradually unroll it beneath him, little by little, working slowly and smoothly, until the victim lies on the fully opened blanket. If the person with a neck injury lies face down, the head should be steadied, the arms brought above the head, and the body then rolled slowly over on to the blanket, which has been stretched out beside him. The arms may then be brought to the sides. In this maneuver, of course, care must be exercised to keep the head, trunk and extremities in line and not allow the neck to be twisted or bent.

If the victim has a broken back and lies face up, he may be rolled over onto the open blanket in the same way as described for a rigid support.

If, however, he lies face down, he may be lifted by two assistants, one grasping him beneath the arms and the other around the thighs just above the knees, and placed gently, still face down, on the open blanket.

Once on the blanket, he may be lifted and carried between two assistants, who firmly grip the blanket on each side of the victim at the level of his shoulders and the middle of the thighs. In the event of neck fracture, head support must be maintained by a third assistant throughout transportation.

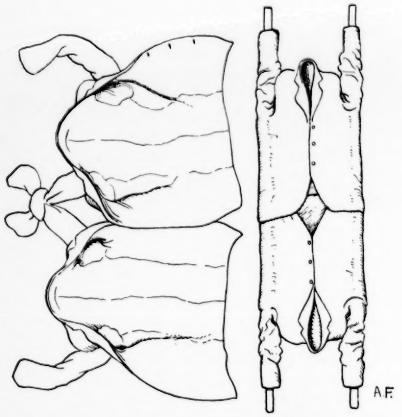

FIGURE 7. *Two Methods of Improvising an Emergency Stretcher with Coats or Jackets.*

It happens sometimes that not even a robe or blanket can be found. Improvisation may still be possible. If two coats or jackets are stretched out side by side, one is buttoned to the other, and the ends of the adjacent sleeves are tied together, a suitable blanketlike support will have been made (Fig. 7). Better still, if two long poles or saplings can be found, a stretcher may be improvised with the two coats as follows. The open coats are stretched out on the ground, tail to tail. Each pole is slipped through the sleeve of one coat and the corresponding sleeve of the other, and each coat is buttoned. This provides a very serviceable stretcher by which two people, grasping the ends of the poles, can transport even a heavy person.

* * *

In conclusion, physicians who are teaching first-aid classes must always bear in mind the fact that the listeners are not actual or potential physicians. Thus, too much knowledge on the layman's part is not taken for granted, simple terms and language that they are sure to understand will be used, and the scope of instruction will be restricted to matters intimately concerned with first aid for fractures. About all, one should stress the points that, whenever possible, medical assistance should immediately be summoned and that the instruction given is for the sole purpose of protecting the victim from further injury until he can receive professional care.

The foregoing suggestions have been made in the hope of lightening to some extent the burden assumed by the physician who volunteers to instruct laymen in emergency splinting. The result of personal experience in this type of instruction, these methods have been tested and found practical and effective. They are not represented as necessarily the best, certainly not the only, procedures that may be followed. But, if adopted, they will be found simple to teach and reliable in application.

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THE EFFECT OF POSTOPERATIVE INTERCOSTAL-NERVE BLOCK ON PULMONARY VENTILATION*

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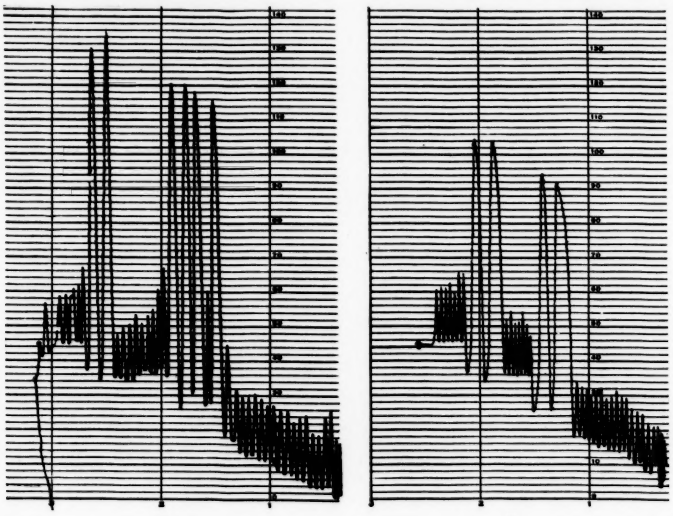
BOSTON

A SIGNIFICANT decrease of pulmonary ventilation following upper abdominal incisions has been repeatedly demonstrated.[1, 2] Figure 1*A* is a spirometric record obtained with a Collins metabolism apparatus on a forty-six-year-old woman breathing air. From this tracing, it may be calculated that the tidal air is 400 cc., the respiratory rate 18 per minute, the vital capacity 1800 cc. and the minute pulmonary volume 7200 cc. Figure 1*B* is a tracing from the same patient, taken in a like manner, twenty-four hours after choledochostomy had been done through a vertical right rectus-splitting incision: the tidal air measured 280 cc., the respiratory rate 22 per minute, the vital capacity 1400 cc., and the minute pulmonary volume 6160 cc. This represents a postoperative reduction in vital capacity of 20 per cent, and in minute pulmonary volume of 15 per cent. A reduction in pulmonary ventilation is the usual change following upper abdominal incisions in women. Beecher[2] found an even greater reduction in vital capacity in men following upper abdominal operations.



A B

FIGURE 1. *Spirometric Records before and after Choledochostomy.*

	A	B
Tidal air	*400 cc.*	*280 cc.*
Respiratory rate	*18*	*22*
Vital capacity	*1800 cc.*	*1400 cc.*
Minute pulmonary volume	*7200 cc.*	*6160 cc.*

Reduced pulmonary ventilation has been ascribed to inhibition of respiratory excursion, to tight dressings, which restrict the costal margins, to splinting of the abdominal musculature resulting from pain in the wound, and to decreased diphragmatic excursion caused by intestinal dis-

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tention. Expansion of the lower lobes of the lungs follows closely the movements of the sixth, seventh, eighth, ninth and tenth ribs and their attached muscles. The diaphragm is the dominant organ in facilitating ventilation of the lower lobes, as Keith[3] has shown. The lower sets of ribs are especially adapted to act with the diaphragm and are an intrinsic part of the diaphragmatic mechanism. Since the oblique abdominal muscles are antagonists to the diaphragm, the spasm induced in them by upper abdominal incision prevents full excursion of the diaphragm as well as of the lower ribs. These factors are involved in the interference with normal pulmonary ventilation and, presumably, are the basis for the occurrence of postoperative pulmonary complications, especially in the lower lobes.

Relief from pain in upper abdominal incisions, by relaxing the reflex spasm of the somatic musculature involved, should therefore improve pulmonary ventilation. Capelle[4] relieved painful spasm in the wound by continuous novocain infusion and reported a resulting improved vital capacity and thoracic excursion. This procedure is impracticable because of the danger of toxicity from the continued administration of the anesthetic as well as interference with wound healing. Collins[5] found an increase in vital capacity following infiltration of the wound with an aqueous solution of Eucupin and novocain. Zollinger[6] injected an almond oil solution of Eucupin base, ethylaminobenzoate and benzyl alcohol into the intercostal nerves of 15 patients who had had upper abdominal operations. By comparing the resulting average vital capacity with the postoperative average determined previously by Cutler and Hoerr,[7] he concluded that the decreased pain of upper abdominal incisions following intercostal-nerve block increased vital capacity. On the basis of 2 favorable cases, Gius[8] recommended paravertebral novocain injection into the intercostal nerves for the improvement of respiratory function in patients with postoperative atelectasis. Since this procedure could cause intercostal-muscle paralysis in addition to relieving pain, the injection of sensory and motor branches to the upper abdomen only, according to Bartlett's[9] technic, appears to be more logical.

This report deals with the effect on pulmonary ventilation of relief of the postoperative wound pain of upper abdominal incisions. Spirometric tracings were made twenty-four hours postoperatively before and after intercostal anesthesia. The Bartlett technic was used. The sixth to the tenth intercostal nerves of 8 patients who had had operations on the biliary tract were injected in the mid-axillary line on the right side to desensitize the upper abdominal wall without affecting the intercostal muscles, whose nerve supply leaves the main intercostal trunk posterior to the midaxillary region.

The anesthetic solution used in this study was 0.2 per cent Eucupindihydrochloride and 1 per cent procaine hydrochloride in Ringer's solution. Although this solution had been used in patients by Collins, its possible damaging effect on various tissues and cavities was first tested by us in rabbits, and no harmful effect was observed. Following intercostal-nerve block, anesthesia persisted for three or four hours, and analgesia remained for more than twenty-four hours; when abdominal pain returned, it was mild in every case. Longer acting agents, such as oily solutions of anesthetic substances, might be more desirable, but since Duncan[10] observed serious degeneration in the motor nerves of the cat following the use of these anesthetic mixtures, they should be applied with caution.

Table 1 summarizes the data on pulmonary ventilation in a group of respiratory tracings obtained before and after intercostal-nerve block twenty-four hours after an upper abdominal procedure. Following relief of upper-abdominal-wound pain by intercostal-nerve block, the respiratory rate shows

TABLE 1. *Effect of Unilateral Intercostal-Nerve* Block on Pulmonary Ventilation.*

Case No.	Respiratory Rate		Tidal Air		Minute Pulmonary Volume		Vital Capacity		Increase in Vital Capacity
	Before Block	After Block	Before Block	After Block	Before Block	After Block	Before Block	After Block	
	per min.	*per min.*	*cc.*	*cc.*	*cc.*	*cc.*	*cc.*	*cc.*	*%*
1	30	25	240	200	7200	5000	880	1120	27
2	15	16	240	280	3600	4480	880	1080	23
3	24	22	480	520	11,520	11,440	1360	1520	12
4	25	25	280	280	7000	7000	1040	1200	15
5	25	35	280	280	7000	9800	580	760	31
6	30	30	280	280	8400	8400	480	600	25
7	23	25	280	320	6440	8000	520	680	31
8†	18	18	280	280	5000	5000	640	640	0
								Average	23

*Nerves of sixth to tenth dorsal vertebras.
†Block partially effective; ? gastric dilatation.

no constant change; in 3 cases, the rate was decreased, in 2 it was increased, and in 3 it was not altered. The tidal air likewise showed no consistent change: in 1 case it was decreased, in 3 it was increased, and in 4 it was unchanged. The vital capacity showed a moderate increase in 7 of the 8 cases following relief of wound pain. In Case 8, the block was only partially effective, and there was some gastric dilatation, which may account for the lack of increase in vital capacity. The average increase of vital capacity in this group of cases was 23 per cent. The minute pulmonary volume showed no constant change, as one might expect, since the respiratory rate and tidal air were not altered in any constant direction.

Although the increase in vital capacity following relief of wound pain by intercostal-nerve block was of itself significant, other benefits were noted. The patients moved about more readily. Coughing was not only easier but also more effective. In 1 striking case, a patient who had high fever, rapid pulse and evidence of consolidation at the lung bases for forty-eight hours after epigastric herniorrhaphy obtained rapid relief of pulmonary signs and symptoms and became afebrile twelve hours after intercostal-nerve block, which effectively relieved the wound pain. This improvement was almost certainly due to the facts that cough became easier and that the patient was able to raise a large quantity of thick sputum. Although relief might have occurred in spite of nerve block, the patient's immediate appreciation of increased comfort in breathing and in coughing was very definite.

Conclusion

The relief of upper-abdominal-wound pain by intercostal-nerve block improves the vital capacity and benefits the patient in other respects. The procedure is therefore worthy of more frequent application.

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MEDICAL PROGRESS

ARTERIAL HYPERTENSION*

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BOSTON

In the last two years, a tremendous amount of work has been done in an attempt to solve the problems of arterial hypertension. Although much has been learned concerning the mechanisms of the renal types of hypertension produced experimentally in animals, and also of their clinical counterparts occurring naturally in man, little of practical value has been added to the methods of preventing or relieving the disease in human beings.

Reprints of articles in this series are not available for distribution, but the articles will be published in book form. The current volume is *Medical Progress: Annual, 1940* (Springfield, Illinois: Charles C Thomas Company, 1941. $4.00).

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Hypertension of Renal Origin

Animal Experiments

Pathologic physiology. It seems clear that the arterial hypertension produced in animals by interference with the blood supply of a kidney[1, 2] is due to the increased pressor activity of the blood leaving that kidney (Fig. 1).[3-6] Furthermore, it appears that this hypertension may be moderated by the presence of a normal kidney,[7, 8] which can reduce the pressor properties of blood that has passed through the so-called "ischemic" kidney.[6] Although the procedures used to produce this type of hypertension may be severe enough actually to reduce the blood flow through the kidney, they may be so nicely adjusted that they are exactly balanced by the ensuing responses in renal hemodynamics and systemic arterial pressure, and thus result in no significant change in renal blood flow.[9, 10]

Strictly speaking, therefore, it may be inaccurate to apply the term, "renal ischemia," to the circulatory disturbance produced in the kidney. It has been shown that the reduction of arterial pulse pressure within the kidney is probably the abnormality that initiates and maintains the renal type of experimental hypertension.[11]

The hypertension produced in animals by clamping of the aorta just above the renal arteries (simulating coarctation) seems to be of the renal

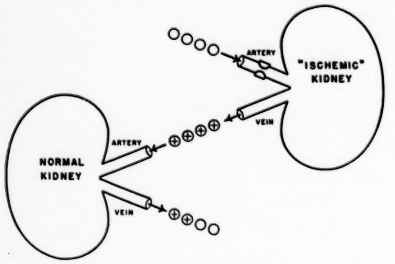

FIGURE 1. *Diagram of the Probable Mechanism of Renal ("Ischemic") Hypertension.*

The blood (circles), on passing through the "ischemic" kidney (with a clamp on the artery), acquires increased pressor (+) activity but, after passing through a normal kidney, loses some of this activity.

type.[6, 12, 13] Intermittent occlusion of the renal artery, up to thirty minutes daily, does not cause a persistent elevation of arterial pressure,[14] although there may be a brief rise of pressure immediately after the release of the occlusion.[15–17]

In animals with renal hypertension, more refined methods of measuring renal hemodynamics[18, 19] have revealed evidence of vasoconstriction of the efferent arterioles of the glomeruli (Fig. 2).[6, 10, 20]

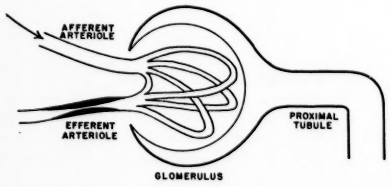

FIGURE 2. *Diagram of a Glomerulus, Showing Constriction of Efferent Arteriole.*

This efferent arteriolar constriction within the kidney itself is apparently part of the generalized vasoconstrictor response to the humoral pressor material elaborated by the ischemic kidney.

Searches for the humoral pressor substance believed to be responsible for renal hypertension have proceeded in two directions: study of extracts of kidney, and study of hypertensive blood.

From the renal cortex, a protein extract may be obtained that, when injected intravenously into intact animals, produces hypertension similar to renal hypertension. Actually, this extract, renin,[21] is not itself a pressor agent, but reacts with a pseudoglobulin fraction of blood (called "renin-activator"[22] or "hypertensin-precursor"[23]) to form a strongly pressor substance, called "angiotonin"[24] or "hypertensin,"[25] which, when injected intravenously, causes arterial hypertension and vasoconstriction generally,[26] including the efferent arterioles of the glomeruli.[27]

Studies of the renal and peripheral venous blood of animals with renal hypertension have shown it to be more vasoconstrictor and vasopressor than normal.[5,6, 25, 28, 29] Treated chemically, such blood may yield material similar to renin, and to angiotonin or hypertensin. In addition, it may exhibit other properties differing from those of renin or angiotonin.[6, 30, 31] However, the increased pressor activity of hypertensive blood has yet to be identified with the presence of one or more specific chemical compounds in abnormal amounts. That a whole series of such compounds is at least theoretically possible is indicated by the observations that similar substances are formed by the action of pepsin on renin-activator (hypertensin-precursor)[32, 33] and of anoxic kidney tissue (decarboxylating enzymes) on amino acids.[34]

Treatment. It was shown early that experimental hypertension due to unilateral renal ischemia could be relieved by removal of the involved kidney.[3] This fact settled a very important question, namely, whether hypertension is necessarily an irversible state. The demonstration that renal hypertension, at least early in its course, is a functional disease, capable of relief, gave a hopeful outlook. This line of work has been extended, however, to demonstrate that, if a severe hypertension is allowed to persist, hypertensive vascular lesions may appear in widespread areas, including the presumably normal kidney.[35–37] Furthermore, removal of the ischemic kidney at this stage may be followed not by complete relief but by a residual hypertension.[36, 38] Thus, it appears that a vicious circle may be established in which the hypertension due to impairment of renal circulation may produce lesions that cause further impairment and, as a result, more hypertension (Fig. 3). The occurrence of malignant hypertension may be due to such a mechanism.[36]

Since it was established that the normal kidney is able to decrease the pressor activity of renal hy-

pertensive blood, it was logical to search for a neutralizing or antipressor substance in the kidney. Renal extracts have been obtained that lower the arterial pressure of hypertensive but not of normal animals,[39–41] and decrease efferent arteriolar constriction.[42] They also neutralize the effects of angiotonin.[42, 43] Similar results have been obtained with tyrosinase[44] and amineoxidase,[45, 46] which were used with the idea of destroying whole groups of pressor substances, if present. Unfortunately, these depressor materials have not yet been freed of toxic and pyrogenic properties, especially when given parenterally. Therefore, caution must be used in attributing their depressor action to the presence of specific neutralizing substances, since it has been shown that various nonspecific vasodilating or pyrogenic materials may have similar effects.[47, 48]

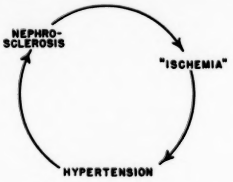

FIGURE 3. *Diagram of the Vicious Circle in Renal Hypertension.*

Clinical Application in Human Beings

Parallel observations have been made in human beings confirming almost every important point learned about renal hypertension in experimental animals. Many cases of so-called "renal hypertension" in man have been reported,[49–51] including a number that have been relieved by removal of a unilaterally involved kidney.[50–55] In children, such cases have been particularly dramatic.[56–59]

Studies of the renal hemodynamics in hypertensive patients have revealed evidence of efferent arteriolar constriction in the kidneys.[48, 60] In those whose arterial pressure has been lowered following the removal of a diseased kidney, the efferent arteriolar constriction in the remaining kidney has been found to decrease.[61] The total renal blood flow in unselected hypertensive patients is usually below normal.[48, 60] However, differential function tests of the two kidneys in such patients have failed to reveal unilateral renal ischemia as a frequent finding in essential hypertension.[62]

Clinical[63] and pathological[53, 64] studies of cases of renal hypertension not relieved, or only partially relieved, by nephrectomy support the view that hypertension of some duration is accompanied by the development of hypertensive vascular lesions, which may involve the normal kidney and thus produce a vicious circle (Fig. 3).[36,65] Structural changes have been found in the arterioles of muscles after renal hypertension of only a few months' duration.[66] Such lesions are indistinguishable from those found in essential hypertension.

Patients with coarctation of the aorta have been shown to have generalized diastolic hypertension,[67] and also efferent renal arteriolar constriction.[68] These observations are in accord with experimental studies suggesting that the arterial hypertension associated with coarctation is renal in origin.

The effects of renin,[69] angiotonin[70–72] and hypertensin[73] have been studied in human beings. Injected intravenously, these substances produce elevations of arterial pressure roughly proportional to the dosage used. The resultant hypertension is similar in many ways to clinical essential hypertension, but differs from it in certain respects, particularly in being accompanied by a considerable rise in venous pressure. Angiotonin produces efferent arteriolar constriction in normal human kidneys.[20, 48, 72]

The use of depressor substances in the treatment of patients with hypertension has been restricted by the scarcity of the materials, and also by the toxic side reactions produced by certain preparations. In some patients, depressor renal extracts are said to lower the arterial pressure, decrease the efferent arteriolar constriction, and relieve the symptoms, especially headache and impairment of vision.[30, 42] However, the effects are not consistent, and are complicated by the unpredictable variations of the disease, as well as by the occurrence of toxic reactions. Until standard preparations can be produced in adequate amounts for general trial, the value of such depressor substances cannot be finally determined.

NEUROGENIC HYPERTENSION

Hypertension of sympathetic nervous origin produced in animals by section of the moderator nerves[74, 75] does not require the presence of the kidneys.[76] Hypertension due to increasing the intracranial pressure of animals apparently depends on reflex sympathetic stimulation of the heart, either directly through the cardiac nerves or indirectly through the mediation of the adrenal glands.[77]

The role of the sympathetic nervous system in human hypertension remains obscure. Elevations of arterial pressure, especially in response to unpleasant stimuli, presumably occur in both normal and hypertensive persons through the mediation of the sympathetic nervous system. However, there is no direct relation between the amount of the

rise in arterial pressure and the degree of sympathetic vasoconstriction in the skin,[78] or in the muscular parts of the extremities[79] during such stimuli. Neither is there a loss of the pressor response after extensive splanchnic sympathectomy.[80, 81] As yet, the sympathetic nervous mechanism in such responses is not clear. It appears that excessive responses (hyper-reactions) are commoner among hypertensive and prehypertensive patients than among persons who never develop hypertension.[82] In fact, it seems that a tendency to abnormal elevations of arterial pressure is more important than the presence of renal disease in determining the subsequent development of sustained hypertension.[83] However, it should be noted that some hypertensive, hyper-reactive patients who have been relieved of their hypertension by the removal of an ischemic kidney have also been relieved of their hyper-reactivity.[84]

A recent progress report[85] has summarized the knowledge to date concerning surgery of the autonomic nervous system in man. In some patients with essential hypertension, extensive sympathectomy definitely lowers the arterial pressure, especially with the patient in the upright position. It apparently does not increase the renal blood flow or decrease the efferent arteriolar constriction.[86–88] Nor does it increase the blood flow in the calf.[79, 80] Sympathectomy, therefore, appears to be more a symptomatic than a specific form of therapy for hypertension. However, one may argue that lowering arterial pressure by any means impedes the development of hypertensive vascular lesions, especially in the kidneys, that might establish a vicious circle. Further long-term studies[89] of the patients already operated on should eventually answer the question of whether sufficient, lasting beneficial results are obtained with surgical sympathectomy to warrant its use in arterial hypertension.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 28301

PRESENTATION OF CASE

A fifteen-year-old schoolboy was admitted to the hospital because of headache and swelling and redness of the face of two days' duration.

For about four months prior to entry, the patient had frequent colds with considerable nasal discharge and postnasal drip. Four days before entry, the patient went swimming in a pool for the first time in several weeks. Two days later he developed a cold associated with severe right frontal headache. The next day the temperature ranged from 101 to 102°F. His headache persisted, and there was swelling of his right periorbital region to such an extent that he could not see out of the right eye. He had no chills, earache or sore throat.

The past and family histories were noncontributory. A year before entry, the patient had had an attack of frontal sinusitis.

On admission, the patient appeared acutely ill. The right eye was partially closed by swelling of the orbital tissues. There was tenderness over the right frontal sinus, with accompanying swelling and redness. Upward gaze was painful. Both frontal sinuses transilluminated poorly. Physical examination was otherwise negative.

The blood pressure was 140 systolic, 80 diastolic. The temperature was 99.4°F., the pulse 72, and the respirations 17.

Examination of the blood showed a white-cell count of 22,200 with 89 per cent polymorphonuclears, and a red-cell count of 4,690,000 with 15.3 gm. hemoglobin. The urine was normal.

Roentgenograms of the sinuses showed gross thickening of the mucous membrane of the right antrum, with polypoid changes; the sinuses were otherwise clear.

On the third hospital day, the temperature rose to 103°F. and the patient had a series of convulsive seizures in which the head and eyes turned toward the right and then generalized convulsive movements appeared, which were more marked in the right arm and leg than in those on the left. Between seizures, the patient was stuporous and showed right hemiparesis, with extensor plantar response on the right, and marked stiffness of the neck. A lumbar puncture gave clear colorless fluid under an initial pressure of 230 mm. of water; the fluid contained 12 polymorphonuclears, 7 lymphocytes and 2 large mononuclears per cubic millimeter. The total protein was 58 mg. per 100 cc., the sugar 103 mg., the chlorides 690 mg., and the gold-sol curve 0012311000.

The seizures increased in frequency until they occurred about every five minutes. The patient was taken to the operating room, but because of severe respiratory difficulty, operation was deferred. The convulsions were controlled by ether anesthesia. Sulfapyridine and heparin were administered. The patient regained consciousness, but had right hemiplegia and aphasia. Two days later, the headache increased and the patient became less responsive. *Staphylococcus albus* was obtained from a blood culture taken on the second hospital day. On the sixth hospital day, an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. FRANCIS C. GRANT*: We have here a child with obvious infection of the frontal sinuses, predominantly on the right side. On the third hospital day, a critical change in his condition developed, with an increase in temperature and convulsive seizures, which began with turning of the head and eyes to the right. These convulsive seizures were much more marked in the right arm and leg than in those on the left, whereas it was the right frontal sinus that was involved. He was stuporous and had a right hemiparesis and a Babinski sign on the right. The convulsive attacks were finally controlled, and the patient regained consciousness but had a right hemiplegia and aphasia. I presume that he was right handed.

DR. CHARLES S. KUBIK: Yes.

DR. GRANT: He must obviously have had a lesion of some type in the left cerebral hemisphere, with the possibility, of course, that, since this was an infectious process and since the sinuses were involved, there might have been multiple infectious processes.

I see nothing here about the eye grounds.

DR. KUBIK: They were normal.

DR. GRANT: I suppose he was too stuporous at any time to observe the visual fields.

DR. KUBIK: No; they were normal. He was not too stuporous for that at times.

DR. GRANT: There is nothing here about the conjunctiva of the right eye. Was that inflamed, and was there any ecchymosis?

DR. KUBIK: There was no ecchymosis, but quite a bit of edema.

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DR. GRANT: There was pain on upward movement of the eye, but apparently no difficulty in the movement of the eye.

DR. KUBIK: No; and no diplopia.

DR. GRANT: Are any x-ray films available?

DR. KUBIK: No; the x-ray films cannot be found.

DR. GRANT: Of course in a child of this age, one would not expect to find calcification of the pineal gland, but one might pick up calcification in the choroid plexus, and any change in its position might be of significance.

It seems to me that there are two possibilities In the first place, we have the story that, four months prior to entry, the patient had frequent colds, nasal discharge and postnasal drip. Then he had this acute crisis, with infection of the sinuses, and probably a blood-stream infection, because of the finding of *Staphylococcus albus* in the blood. My suspicion is that the acute frontal sinusitis may not have been the important factor, but only a part of the picture that drove him into the hospital. He might conceivably have had a lesion resulting from the earlier infection, such as an abscess well forward in the left frontal area, that preceded the development of the right frontal sinusitis. The convulsive attacks are certainly suggestive of a left-sided lesion, although convulsive seizures are not a common occurrence in a subcortical abscess. It is quite conceivable that he had thrombosis of the longitudinal sinus. I imagine that this thought occurred to someone else, because the patient was given heparin during his hospital stay.

Am I allowed to elaborate on the way this case should have been handled, on the sixth hospital day, when an operation was performed?

DR. TRACY B. MALLORY: Yes.

DR. GRANT: Presuming that the patient's situation had improved and that the condition for which the heparin and sulfapyridine had been given had cleared up, I should have tapped the ventricles. If, as I suspect, the right ventricle was found to be larger than the left, I should have taken this as evidence of a lesion in the left frontal area and tapped there, expecting to find pus either on the surface or in the brain.

A PHYSICIAN: Is it likely that he had osteomyelitis?

DR. KUBIK: X-ray films taken with that in mind did not show an osteomyelitis.

A PHYSICIAN: Would you consider a subdural empyema?

DR. GRANT: I considered it, and I presume, if present, it would have been in the left temporal region, where it would possibly be picked up when one tapped for abscess.

DR. JAMES C. WHITE: Would you wait for the x-ray films to be positive before making a diagnosis of osteomyelitis?

DR. GRANT: No, I should not wait for them. I am presuming the physicians in charge nursed the patient for the respiratory difficulty he was in, and considered that if he ever was going to be operated on, he should be operated on then.

A PHYSICIAN: Do you think he had a right orbital abscess?

DR. GRANT: They did not do anything about it if he had. I take it that the orbital swelling disappeared. Did it disappear?

DR. KUBIK: No, it did not disappear; but it did not get any worse, and the patient was not operated on for orbital abscess.

A PHYSICIAN: Why do you lean to temporal-lobe abscess instead of frontal?

DR. GRANT: I am presuming that the boy had some difficulty with speech. They state that aphasia developed. I do not believe one can tell whether the effect of the lesion on the speech center was from in front or from behind or whether it lay directly in the speech center. I think we have to presume that the infection started in the sinuses in front. The lesion is more likely to be frontotemporal than temporoparietal.

DR. WILDER PENFIELD: How can you tell irregularity of the ventricles?

DR. GRANT: You can find the difference in size between them by the amount of fluid withdrawn from each of them.

DR. PENFIELD: I wish I could.

A PHYSICIAN: Why do you object to putting air in?

DR. GRANT: I think this child was too sick. A ventricular estimation is sufficient.

A PHYSICIAN: Would you consider the injection of Thorotrast?

DR. GRANT: No, I think this boy was too sick. He had been carried through a respiratory collapse. The respiratory collapse is commoner with a cerebellar than with a frontal lesion. I do not have any data here regarding what immediately preceded the severe respiratory difficulty. Was there an accompanying drop in pulse, or was it purely respiratory? Was it due to positional change on the operating table or to the administration of anesthesia?

DR. KUBIK: It occurred before the anesthesia was started. He began to have seizures, almost constantly, and then stopped breathing. They telephoned from the operating room, as a matter of fact, that the patient had died.

A PHYSICIAN: Is there any relation between the trouble and the spinal puncture?

DR. GRANT: I should presume that they would not remove any amount of fluid — just a drop or two to get the cell count.

A PHYSICIAN: They did the total protein and chloride.

DR. GRANT: That requires about 8 cc. I should not have done that. The record does not state what the sequence was. It may have been done some time before.

A PHYSICIAN: The convulsions were very frequent for an abscess, were they not?

DR. GRANT: Yes; my conception about this boy is that he had a frontal abscess before the acute symptoms developed. Then sinus thrombosis produced cortical infection, irritation and convulsions.

A PHYSICIAN: Would you consider that the aphasia and hemiplegia were purely postconvulsive?

DR. GRANT: No, because they came on, as I gather, late. I think you are correct in presuming they might have been. It simply says that the patient regained consciousness, then had aphasia and right hemiplegia.

A PHYSICIAN: What is wrong with the diagnosis of acute early encephalitis deep in the frontal lobe?

DR. GRANT: The cell count was not particularly suggestive of encephalitis: it was not high enough, nor do I believe that the cells would have been predominantly polymorphonuclears. It looks more like abscess.

CLINICAL DIAGNOSIS

Subdural abscess, left motor area.

DR. GRANT'S DIAGNOSES

Left frontal abscess.
Subcortical longitudinal sinus thrombosis, spreading to veins over left cortex and producing patchy meningitis and convulsive seizures.

ANATOMICAL DIAGNOSES

Subdural empyema.
Thrombophlebitis of superior longitudinal sinus.
Frontal sinusitis.
Frontal osteomyelitis.

DR. KUBIK: This patient was sent up to the operating room the first time with a diagnosis of subdural abscess, but because of the respiratory difficulty, the operation was postponed. The seizures were controlled with ether. He regained consciousness, but the hemiplegia and aphasia persisted. Three days later, he became worse, and a left subdural empyema was drained through a lateral frontal burr hole. The patient had a very stormy convalescence, with a number of convulsive seizures, but he finally pulled through and recovered completely from the aphasia and hemiplegia. In the two years following his illness, he has had three or four seizures, and he is now taking dilantin. There was no x-ray evidence of osteomyelitis at the time of operation or for two or three weeks after that. Then we stopped taking x-ray films, and he was discharged after about seven weeks. Two weeks later, he came back with frontal osteomyelitis, demonstrated by x-ray study. This was operated on, and at the operation, the superior longitudinal sinus was found to be thrombosed. It seems likely that the thrombophlebitis preceded and was responsible for the subdural injection.

CASE 28302

PRESENTATION OF CASE

A fifty-five-year-old Canadian toolmaker was admitted to the hospital because of nausea, diplopia, vertigo, headache and spells of unconsciousness and disorientation occurring over a period of at least six weeks.

The patient had suffered from "migraine headaches" most of his life. These appeared about once a month and were accompanied by scotomas, nausea and vomiting. Four years before entry, the patient was in an automobile accident. He struck the left side of his head and was unconscious for a few minutes. Subsequently, there was insidious appearance of irritability, lack of judgment, memory loss and disorientation. These personality changes became progressively more marked. One year before entry, the usual headaches changed character, becoming occipital, with pounding radiation to both frontal regions and associated nausea, vomiting and vertigo. Eight months before entry cholecystectomy was performed at a community hospital; "stones" were found. Six weeks before entry, there was marked increase in the nausea and vomiting that accompanied the headaches. The patient went to a community hospital, where "an old duodenal ulcer" was demonstrated roentgenographically; treatment by the Sippy diet afforded no relief. After three weeks, the patient was transferred to another community hospital. The headaches continued as before, and the patient suddenly became comatose during the course of one. A lumbar puncture showed an initial spinal-fluid pressure equivalent to between 350 and 375 mm. of water. His pulse rose from 40 to 100 following the puncture, and there was temporary relief of symptoms. He was then transferred to this hospital.

On admission the patient was disoriented, vomiting and complaining of dizziness and double vision. There was evidence of recent weight loss and dehydration. The heart, lungs and abdomen

seemed normal. The neck was stiff, and there was a Kernig sign. The pupils were unequal, alternately enlarging and contracting. The optic disks were normal. There were questionable hypesthesia and hypalgesia in the left trigeminal-nerve field. The musculature of the trunk and extremities was generally wasted and flaccid, possibly more so on the left than on the right. The tendon reflexes were hypoactive and equal.

The blood pressure was 120 systolic, 80 diastolic. The temperature was 99°F., the pulse 44, and the respirations 15.

Examination of the blood showed a white-cell count of 16,700 with 71 per cent polymorphonuclears, and a red-cell count of 4,900,000 with 85 per cent hemoglobin. The urine showed a ++ test for albumin.

Roentgenograms of the skull showed no abnormalities. A roentgenogram of the chest showed only a few small areas of calcification in the right lung.

The patient became increasingly stuporous. On the second hospital day, bilateral occipital burr holes were made, under local anesthesia. Clear colorless fluid was obtained from both ventricles. The pressure was considerably increased, particularly on the left. The fluid from the left ventricle showed 34 polymorphonuclears and 12 lymphocytes per cubic millimeter with a total protein of 36 mg. per 100 cc. The fluid from the right ventricle showed 62 polymorphonuclears and 28 lymphocytes per cubic millimeter with a total protein of 44 mg. per 100 cc. The gold-sol curve was 0001210000, and the Wassermann reaction was negative.

Following the tap, the patient seemed improved. Adequate hydration was obtained by vigorous intravenous therapy. Ventricular tap was repeated on the second night in the hospital because of the return of drowsiness, disorientation and a slow pulse, with spasticity of the left arm and leg. Twelve cubic centimeters of orange fluid was aspirated, with improvement in the patient's condition. During the next few days, much restlessness necessitated paraldehyde sedation. A right hemiparesis appeared. An electroencephalogram suggested "electrical pathology" in the right frontal lobe. A ventriculogram performed on the third hospital day showed slight dilatation of the lateral ventricles, more marked on the left, and slight widening of the third ventricle; the surface filling showed some widening of the sulci. No tumor was demonstrable. On the eighth hospital day, bilateral temporal burr holes were made under local anesthesia, and the subdural space was explored without significant findings. Subsequent lumbar punctures gave xanthochromic fluid, with an initial pressure of 440 mm.

The patient remained restless and unco-operative, constantly pulling off his dressings. Ultimately the left occipital burr hole became septic. On the twenty-third hospital day, the temperature rose to 104°F., and there was patchy bronchial breathing, with moist rales at the base. Sulfapyridine was given, but death occurred on the following day.

Differential Diagnosis

Dr. Jost Michelsen: This is a very complex story, but one conclusion seems to be fairly safe: shortly before and on admission to the hospital, this patient had an intracranial lesion that produced an elevation of intracranial pressure. The findings on lumbar puncture in the outside hospital and on ventricular examination in this hospital bear out that point. Additional evidence can be derived from the facts that, following the lumbar puncture, the pulse rose from the very slow rate (40) to 100 beats per minute and that there was temporary relief of symptoms. In view of these findings and observations, the absence of papilledema is of little significance.

The questions arise, Where in the cranial cavity was this lesion localized, and what type of lesion did this patient have? Let us start with the findings on admission. The patient complained of double vision. We do not know which eye muscles or nerves were involved. Weakness of the sixth, as well as of the third, nerve can be interpreted as general signs of pressure. The pupils were unequal, and there was a hippus. Perhaps these signs, together with the rigidity of the neck and Kernig sign, suggest that there was a tentorial pressure cone, which produced compression of the midbrain. Hypesthesia and hypalgesia of the left trigeminal field may be caused by a lesion anywhere in the course of the trigeminal nerve from the gasserian ganglion to the pons. The flaccidity of all muscles and corresponding sluggishness of the tendon reflexes do not mean very much if we consider the poor general condition of the patient on admission. Later, spasticity of the left arm and leg developed, apparently without weakness. I wonder if the observer made a distinction between spasticity and rigidity. If the change of muscle tension involves both the flexor and extensor muscles, it should be called "rigidity," but if the antigravity muscles are chiefly involved, the condition should be designated by the term "spasticity." Rigidity is found clinically in diseases of the extrapyramidal system; spasticity arises from lesions of the pyramidal system below the level of the cortex or, if of the

cortex itself, from combined involvement of the motor area and the extrapyramidal area.

During the next few days, right hemiparesis appeared. This time the state of the involved muscles is not given. Nor is it mentioned whether the patient became aphasic. Was he right handed or left handed? The electroencephalogram indicated "pathology" in the right frontal lobe. X-ray films of the skull were normal, and hence the pineal gland was in the midline; and the ventriculogram failed to demonstrate a shift or filling defect of the ventricular system. Consequently, we must assume, contrary to the localization by the electroencephalogram, that the process was a diffuse one and certainly not limited to one hemisphere of the brain.

The question of the type of lesion is not less involved. A brain tumor can be ruled out. The absence of changes in the ventriculogram is not compatible with such widespread involvement by tumor. There is no evidence of a primary focus to indicate multiple metastases to the brain: the albumin in the urine is not sufficient evidence, and no other changes of the urinary tract are reported.

Thus, there remain to be considered subacute or chronic, vascular or inflammatory lesions. I say subacute or chronic lesions because it is difficult to determine from the history when this fatal disease started.

The patient had suffered from headaches most of his life. Their periodic occurrence and association with scotomas, nausea and vomiting are indeed suggestive of migraine, but it must be kept in mind that there are other conditions with constant organic pathology that give rise to similar perineuric manifestations first, and reveal their true character later. The commonest lesions of this type are hemangiomas of or near the occipital lobe. This possibility, however, is remote, because a hemangioma should have been shown in the ventriculogram.

The next point that must be settled is the possible relation of the head injury to the lesion that resulted in this man's death. Following the injury, he developed symptoms such as irritability, lack of judgment, memory loss and disorientation. The underlying pathology of such a postconcussional syndrome may be widespread physical damage to the brain, and I assume that the asymmetrical dilatation of the lateral ventricles and the widening of the third ventricle, as well as of the sulci, demonstrate objectively this pathology in the form of atrophy of the brain. But this alone did not account for the signs that the patient developed in the last stage of his disease. He may have had a subdural hematoma in addition to the other damage to his brain. This is a good possibility. The delayed onset of symptoms in subdural hematomas is not unusual, and the clinical picture is so variable that often the diagnosis cannot be made without trephine exploration. Such exploration was carried out on the eighth hospital day, without significant findings. It seems to me that it should have preceded the other operations. One can assume, however, that the patient did not have a subdural hematoma; furthermore, the ventricular puncture performed as the first procedure on the second hospital day yielded valuable information. There was an increase of cells and protein in the ventricular fluid. I am unable to evaluate the difference in pressure, cells and protein on the two sides, but the presence of white cells and the increase of protein in the ventricular fluid are very suggestive of an intracranial infection. Occasionally, brain tumor close to the ventricular system produces similar changes, but brain tumor has been ruled out.

It is unfortunate that the examination of the cerebrospinal fluid obtained in the outside hospital was incomplete, and it is equally unfortunate that the lumbar puncture was not repeated after his admission here, in spite of the beneficial results of the first lumbar tap. I should like to know about the cell count, the sugar, the chlorides and the findings on bacterial examination of the spinal fluid. The examination of the ventricular fluid apparently was also incomplete. The negative Wassermann reaction, however, eliminates a syphilitic infection.

Because of the lack of adequate information concerning the cerebrospinal fluid, the analysis from now on must be based more or less on guesswork. The trauma was not such as to suggest the development of an intracranial abscess or multiple abscesses. There was no evidence of an external compound fracture or cerebrospinal-fluid leak, and there was no other focus from which they could have originated. Did this man have an encephalitis? In reviewing the clinical findings once more, one wonders if rigidity of the neck and the Kernig as well as the other signs were not produced by the intracranial infection rather than by a pressure cone; in addition, signs of meningeal irritation are rare in encephalitis. Tuberculous meningitis may be considered. The negative chest plate does not rule it out. The temperature in tuberculous meningitis is often markedly irregular, which may explain the absence of any elevation in the beginning. A fungous infection of the central nervous system may be quite consistent with the course of the disease in this man. Metastatic carcinomatosis of the meninges is unlikely since, as previously mentioned, no primary focus was found. There is no evidence that the patient had a meningitis originating from the ears or sinuses, which apparently were normal. There was no osteomyelitis of the skull or any other source

from which an infection of the venous system of the brain may have originated. Furthermore, no definite relation can be established between the intracranial process and the abdominal lesions.

In summary, I should say that the patient suffered from migraine, gallstones and a duodenal ulcer. The head injury produced atrophy of the brain. In addition to these changes, the patient had widespread intracranial disease, with increased intracranial pressure and irritation or infection of the meninges. Brain tumor is ruled out. Single or multiple intracranial abscesses are considered unlikely. Metastatic involvement of the brain and meninges is eliminated. Tuberculous and Torula meningitides, as well as encephalitis and other forms of meningitis and thrombophlebitis, are unlikely. In other words, a definite differential diagnosis of the intracranial process in the final stage is impossible. There were terminal bronchopneumonia and wound sepsis.

Clinical Diagnoses

Encephalitis (type undetermined).
Cerebral atrophy.
Terminal bronchopneumonia.

Dr. Michelsen's Diagnoses

Migraine.
Gallstones.
Duodenal ulcer.
Widespread intracranial disease (meningitis? encephalitis?).
Terminal bronchopneumonia.
Wound sepsis.

Anatomical Diagnoses

Carcinoma of stomach, with metastases to meninges, brain and left adrenal gland.
Operative wounds: bilateral burr holes, occipital and temporal; cholecystectomy; and appendectomy.
Subdural abscess, postoperative.
Lateral-sinus thrombosis, acute.
Congenital anomaly: extreme hypoplasia of left kidney, with ureter draining into seminal vesicle.

Pathological Discussion

Dr. Tracy B. Mallory: This patient puzzled the clinicians in charge of this case on the ward quite as much as it has Dr. Michelsen. They were never willing to commit themselves to a definite diagnosis. When the patient first came in there seemed to be a slight though remote chance of subdural hematoma, and the original burr holes were made primarily with the purpose of ruling out this one potentially remediable condition. When nothing was found, the impression shifted somewhat in favor of encephalitis, which was the final diagnosis on the death report.

At autopsy, obvious sepsis of the operative wound was found, which had extended through one of the burr holes to produce a localized subdural abscess. There was also a fresh, nonocclusive thrombosis of the lateral sinus. The meninges elsewhere over the cortex showed injection, and thickening and opacity of the arachnoid were also present. It was assumed that this represented a rather chronic meningitis. In the rest of the examination, numerous lesions were found that did not seem to have any connection with the cerebral process. There was a healed ulcer of the duodenum, and a second, still active, ulcer in the prepyloric area of the stomach. The gall bladder had been removed, and there was some dilatation of the common bile duct and hepatic duct. There were anomalies of the urinary tract. The left kidney could not be identified. The left ureter was completely atretic and emptied into the left seminal vesicle instead of entering the bladder at the trigone. The right kidney showed compensatory hypertrophy. A small tumor nodule was found in the medulla of the left adrenal gland. When the microscopic sections came through, it was at once apparent that the thickened arachnoid was diffusely infiltrated with tumor cells, some of which had a characteristic signet-ring appearance. Examination of the prepyloric ulcer showed its margins to be frankly carcinomatous, and the nodule in the adrenal gland also proved to be a metastasis. Although no gross metastases could be found within the cerebral substance, numerous minute foci of tumor cells were found on microscopic observation.

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SUBDURAL HEMATOMAS

The formation and organization of a blood clot beneath the dura covering the brain, either completely composed of blood or partly mixed with cerebrospinal fluid, has long been recognized as a serious sequela of cerebral trauma, or as an occasional accompaniment of the blood dyscrasias and scurvy. A clear pathological concept of subdural hematomas, an understanding of how commonly the lesion may be found if constantly kept in mind by the clinician and an appreciation of the favorable influence of surgical treatment, when applied early, have been arrived at only in comparatively recent times. In 1933 and 1934, many erroneous ideas concerning subdural hematomas were erased from the record by Leary and his associates, and it is to him that we are indebted for our modern concept of this disease. His work, as Munro points out in this issue of the *Journal*, is both "fundamental and unquestionably accurate." Thanks to Leary and subsequent investigators, these lesions can now be divided into two broad groups — the solid type and the mixed type, the latter in its common chronic stage often forming a fluid cyst, sometimes called a "hydroma" or "hygroma," a descriptive term of no particular significance.

Munro has shown that the solid type, usually thought of as the commonest of all subdural hematomas, is, in his experience as head of a large, active, neurosurgical service, the least common. The solid forms are of the nonexpanding type, some caused by trauma and others due to blood dyscrasias. They are often not diagnosed in their acute stage and only disclose themselves as fortuitous findings at autopsy in patients who, not infrequently, have become psychotic. It is this form of subdural hematoma that has received undue emphasis in textbooks and current medical thought, partly owing to the prominence given it by Putnam and Cushing in 1935. In Munro's series, it formed only 45 out of 310 verified cases. The lesions, however, are often favorably affected by surgical intervention, although the operative mortality may rise to 33 per cent, largely the result of deaths in the older age group.

The mixed forms are commoner than the solid types and are exclusively due to craniocerebral injuries. They consist of a mixture of blood and cerebrospinal fluid and expand during the course of their development. Their growth is often rapid in the first five weeks after injury and then somewhat slower for the subsequent two months. A subvariety, comprising the fluid group, accounts for many of the late manifestations of cerebral injury, including the debilitating post-traumatic neuroses.

The importance of both types lies in early recognition and surgical treatment. Theoretically, no acute subdural hematoma should ever reach the chronic stage. The diagnosis, possibly suspected from the clinical course of the patient, must usually

be confirmed by exploratory trephination, transtemporal by preference and bilateral if necessary. Such an operation, in Munro's experience, is never contraindicated in "a patient who has been knocked unconscious by a blow on the head and who is not getting better or is getting worse under adequate lumbar-puncture and fluid therapy." If, as Munro rightfully concludes, these patients are not progressing favorably, "the responsibility rests with the physician to recognize the possibility that a mistake has been made in the diagnosis and that, as a result, he must investigate and treat the cause of this failure to recover by every means at his command." The "means" is the exploratory trephine, lightly, but not derisively, called "woodpecker surgery." In the hands of those specially skilled to carry it out, such surgical treatment not only prevents serious brain damage but also, in many cases, saves lives.

FIFTIETH ANNIVERSARY OF THE GRENFELL MISSION

On August 4, 1892, a recent graduate of the London hospitals, Wilfred T. Grenfell, landed on the Labrador coast. On that bleak and isolated land, he found British fishermen and trappers who, amid great difficulties, earned a meager living for their families; there he also found poverty and disease unchecked and no physician on over hundreds of miles of coast. Fifty years ago, Grenfell saw and met the challenge, and for the rest of his life, he labored to improve the health and the lot of the fisherfolk of Labrador and northern Newfoundland. Last summer, the ashes of Sir Wilfred Grenfell were buried on the hill above the main mission hospital.

During the years of his active life, the Grenfell Mission, as it is known today, gradually took form, until its work covered over a thousand miles of coast. It grew to comprise five hospitals, as many nursing stations, three schools, a children's home, hospital ships, a marine railway and numerous industrial and agricultural facilities. In spite of his death, and under added difficulties engendered by the war, the work of the mission is being carried on with these tools, under the supervision of a New Englander, Dr. Charles S. Curtis.

By the constant labors of the staff and through the education of the people, the years have witnessed many medical advances. Beriberi and other deficiency diseases, once all too common, have now nearly disappeared. Epidemics of typhoid fever and diphtheria, with their high mortalities, have been abolished by means of mass immunization. Tuberculosis, a very prevalent disease, is being brought under control. But perhaps most important of all, the people of Labrador now recognize the value of the aid extended to them and are willing to go to the nursing stations or hospitals during the early stages of their illnesses.

Today, as in the past,—when over sixty-five physicians from New England alone have given months or years of service,—much of the mission work depends on the continued support and enthusiasm of volunteer and paid workers. To these, Sir Wilfred Grenfell has left a legacy of spirit and courage for service.

MEDICAL EPONYM

Pott's Disease

Caries of the spinal column and its effect were described by Percivall Pott (1713-1788), F.R.S., surgeon to St. Bartholomew's Hospital, in his "Remarks on that kind of Palsy of the Lower Limbs, which is frequently found to Accompany a Curvature of the Spine, and is supposed to be Caused by It: Together with its method of cure." This appears in Volume 3 (pages 349–385) of the edition of his works published in London in 1779.

> The disease of which I mean to speak, is generally called a palsy, as it consists in a total or partial abolition of the power of using, and sometimes of even moving the lower limbs, in consequence, as is generally supposed, of a curvature of some part of the spine. . . . I have, in compliance with custom, called the disease a palsy; but it should be observed, that notwithstanding the lower limbs be rendered almost, or totally useless, yet there are some essential circumstances in which this affection differs from a common nervous palsy: the legs and thighs are, I have just said, rendered unfit for all the purposes of locomotion, and do also lose much of their natural sensibility, but notwithstanding this, they have neither the flabby feel, which a truly paralytic limb has, nor have they that seeming looseness at the joints, nor that total incapacity of resistance, which allows the latter to be twisted in almost all directions; on the contrary, the joints have frequently a considerable degree of stiff-

ness, particularly the ankles, by which stiffness the feet of children are generally pointed downward, and they are prevented from getting them flat upon the ground. . . . I found, in infants, in young children, and in those who had been afflicted with the disorder but a small space of time, that the ligaments connecting the vertebrae, which formed the curve, were in some degree altered from a natural state. . . .

When we attribute the whole of this mischief to the mere accidental curvature of the spine, in consequence of violence, we mistake an effect for a cause; and that previous both to the paralytic state of the legs, and to the alteration of the figure of the back bone there is a predisposing cause of both, consisting in a distempered state of the ligaments and bones, where the curve soon after makes its appearance.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE

CASE HISTORY: SEPARATION OF THE PLACENTA, COMPLICATED BY CHRONIC NEPHRITIS AND HYPERTENSION, WITH DEATH OF THE PATIENT, UNDELIVERED

A thirty-six-year-old multipara, approximately six and a half months pregnant, entered the hospital with acute abdominal pain. Her previous obstetric history revealed eight full-term, normal children, the seventh pregnancy four years earlier having been complicated by toxemia and convulsions; this was followed by a normal pregnancy. There was no history of surgical operations or of scarlet fever, but she was known to have had chronic nephritis and hypertension for some years, and examination of the heart had, for some time, shown the hypertrophy consistent with this combination of diseases.

When she entered the hospital, the blood pressure was 230 systolic, 170 diastolic, there was a definite trace of albumin in the urine, and the sediment contained "casts." Examination of the eyes showed papilledema and signs of recent hemorrhage in the retina. The patient was kept under observation for forty-eight hours; at the end of that time, the acute abdominal pain recurred. Examination of the uterus showed it to be tender and firm. There was no mention of bleeding, but a diagnosis of separated placenta was made. Vaginal examination showed a multiparous cervix admitting two fingers. The patient was treated conservatively by the Dublin method: the cervix and vagina were tightly packed with gauze and a Spanish windlass was applied. Since the patient bled through this pack, a Voorhees bag was introduced into the cervix, the vagina repacked, and a transfusion given. She died undelivered, however, shortly after this.

Comment. From the medical standpoint, of course, this patient should never have been allowed to become pregnant, and in the event of pregnancy, therapeutic abortion was certainly indicated. Any woman with chronic hypertension and nephritis jeopardizes her life when she undertakes pregnancy. This patient received no prenatal care whatsoever, and it is hardly fair to blame the medical profession for the catastrophe. Had she sought medical help earlier in pregnancy, therapeutic abortion should have been advised.

Separation of the placenta as early as the seventh month of pregnancy with complicating nephritis and hypertension is not common but, as evidenced here, quite possible. The blood pressure on entry, the albumin, sediment and casts in the urine, and the eye changes were evidence of very definite kidney damage. The patient was treated ideally — no criticism of her care is warranted. That she should, at this stage of pregnancy, bleed to the point of exsanguination when the cervix and vagina were packed is extremely unusual.

The treatment of this case should have begun with prophylaxis—by the avoidance of pregnancy or by therapeutic abortion and sterilization if pregnancy occurred.

MASSACHUSETTS MEDICAL SERVICE

INCOME LIMITS DETERMINED FOR SUBSCRIBERS

In an effort to create for the low-income employee groups a larger measure of protection than a cash-indemnity contract provides, the Massachusetts Medical Service faces the unwelcome necessity of determining income limits below which medical-service contracts will be sold.

Medical-service contracts refer, of course, to the services that will be provided by participating physicians for surgery, obstetrics and diagnostic x-ray studies. The term is used in contrast to the cash-indemnity, or limited, contract that will be available to individuals in any income bracket above the specified limits. The Massachusetts doctors are initiating their program by offering this medical-service contract only to members of employee groups whose individual income is $2000 or less or whose family income is $2500 or less. That such an arbitrary income limit is fair to both the physicians and the subscribers has been determined after serious study of medical-service programs throughout the country.

The states of California, Oregon, Washington, Michigan, North Carolina and Pennsylvania, the counties surrounding Buffalo and Utica, New York, and the City of Dallas, Texas, have plans well under way. Within recent months, New Jersey and Colorado physicians have established statewide plans. In all these states, the highest level to which the medical-service contract is available ranges from $2000 to $3000.

Decisions in these pioneer states were based on surveys of income levels, on the requirements of state insurance departments and on the need for a sufficiently broad base to render enough individuals eligible to meet the percentage requirements for group enrollment.

In California, physicians set the upper level of eligibility at $3000, the base figure in a comprehensive health-insurance bill that was nearly passed. New Jersey established $1600 as the level for the family, found it too low and raised it to $2500 on the basis of government legislation proposed for the low-income group. Michigan established the limit at $2000 for the individual and $2500 for the family. Because the state insurance department would not accept lower levels, Buffalo set the limits at $1800 for the individual, $2500 for man and wife, and $3000 for the family. Utica has no limit on eligibility.

Basic Figures Result of Study

In addition to the study of the plans operating in other states, the Massachusetts Medical Society has arrived at its $2500 base figure through a study of income groups in Massachusetts. The most normal of recent years was the year 1939-1940, inasmuch as it was free from the abnormal influences of depression or boom. The study revealed that an individual income level of $2000 accounted for 83.4 per cent of the wage earners and that a $2500 family income embraced 91.4 per cent. In the present period of increasing wages, these percentages may be modified. With the increased cost of living, however, individuals within these fixed limits are even more in need of a medical-service program.

Employees whose individual or family incomes place them above the medical-service contract may be enrolled with their group on a cash-indemnity basis. They will receive cash payments for medical services in the amounts specified in their contracts. Since this cash-indemnity subscriber may then be charged by the physician the difference between the latter's fee and the amount guaranteed by the contract, there is virtually no alteration in the present financial arrangement between the private patient and his physician, except that the doctor is assured more prompt payment and the subscriber is enabled to pay his bill more easily.

Summarizing, then, the physicians of the state through the Massachusetts Medical Service are offering medical-service contracts, which comprise surgery, obstetrics and diagnostic x-ray services, to groups of employees whose individual incomes are $2000 or less and whose family incomes are $2500 or less, and cash-indemnity contracts for employees whose incomes are above these figures.

WAR ACTIVITIES

PROCUREMENT AND ASSIGNMENT SERVICE

Duties of the Various Units

The following memorandum concerning the duties of the various units of the Procurement and Assignment Service was recently issued by the executive officer, Major Sam F. Seeley, by order of the Directing Board:

Directing Board

1. Establishment of policies and procedures for the Procurement and Assignment Service.

2. Maintenance of liaison with the appropriate governmental officials and agencies and with the various professional groups.

Central Office

1. Maintain contacts with federal agencies relative to their needs for physicians, dentists and veterinarians, and consultations with these agencies regarding the possibilities of revision of their requests in consideration of the limited supply of professional men in these fields.

2. Prepare quotas of the minimum medical, dental and veterinarian services which should be retained for the civilian population, including private practice, hospital service, industrial service, public-health service and medical education.

3. Prepare quotas for allocating to the states the requests for physicians, dentists and veterinarians needed for war service (these quotas to be determined on the basis of the physician-population ratio in the state, the number of physicians already in service from that state and so forth).

4. Maintain rosters of physicians, dentists and veterinarians:

   a. Total in the United States.

   b. Those who have registered with the Procurement and Assignment Service.

   c. These rosters to contain physician's age, qualifications, location and so forth.

   d. From these rosters, which will be maintained by the National Roster, names of physicians, dentists and veterinarians with certain qualifications will be obtained from time to time.

5. Secure information for the various governmental agencies in regard to physicians, dentists and veterinarians, as to:

   a. Availability for service other than in their present location.

   b. Their professional and other qualifications.

   c. Their willingness to serve in various capacities during the war emergency.

6. On the basis of this information, select the names of those physicians who meet the specifications of the requisitioning agency.

7. Co-operate with the various governmental agencies in obtaining the applications of those physicians thus selected for service.

Chicago Office

1. Maintain and keep up to date the confidential information concerning all physicians, dentists and veterinarians, with respect to character, type of practice, infringements of law and so forth, which must be considered by the Army, Navy and so forth in deciding whether individuals are qualified for commission.

2. Maintain confidential lists of the relative standing of all specialists. (These lists have been developed through the facilities of the American Medical Association, the American Specialty Boards, the National Research Council and confidential advisers representative of the various specialties. This material is to be used by the requisitioning agencies as a basis for assignment of those now on duty and those who are [illegible] for commission.)

3. Furnish assistance of a consultative and advisory nature to the Directing Board and to the various committees of the Procurement and Assignment Service. (This includes the utilization of statistical data collected over a period of many years by the medical, dental and veterinary medical associations.)

Corps-Area Committees

1. Supervise the work of the state committees in order that they will be reasonably uniform in the manner in which they carry out the policies of the Directing Board. (This will require meetings of the corps-area committees with state chairmen and visits by the corps-area chairman to the states within his [illegible] area.)

2. Act as an appeal board in cases in which [illegible] individual physician, dentist or veterinarian, his committee or his employing agency differs with the classification given by the state committee.

State Committees

1. Obtain the overall enrollment of the professional in the state. (This will require the maintenance of rosters in the state offices of those who have enrolled with the Procurement and Assignment Service and those who have not. The former lists will be obtained from the Central Office.)

2. Survey local needs for professional services in conformity with the policies laid down by the Directing Board, and on the basis of these surveys, determine how many physicians, dentists or veterinarians are needed in the various communities of the states to care for the civilian needs and how many can be released for service elsewhere.

3. Determine which particular individual physicians, dentists or veterinarians can be considered "available" for service elsewhere. (In view of the changing circumstances, this will require constant reappraisal and obviously can be done only locally.)

4. Pass on the availability, character and professional qualifications of individual physicians who are being considered for appointment for service elsewhere, for example, for commissions in the Army, Navy and so forth, and

5. Co-operate with the state offices of the Selective Service System in determining whether physicians, dentists and veterinarians who are subject to classification by the Selective Service System are essential in their local communities.

6. Maintain lists to be transmitted from the Central Office of those who have expressed their preference for service in industrial practice, civil practice in other communities, state and local health departments and institutions, and act as liaison between these individuals and the industrial organizations, civil practitioners and health departments and institutions desiring the services of these individuals in a temporary capacity for the duration of the war.

7. Keep the Directing Board informed of conditions in the state, and bring to its attention matters which may involve general policies.

8. Make periodical (weekly) reports to the Central Office of the names and addresses of those commissioned from the state, with information as to which of these have been serving as interns or residents.

District or County Committees

1. Provide information, assistance and advice to the state committees in carrying out their functions. (Local committees have no authority to make final decisions whether positions or individuals are essential or nonessential.)

MISCELLANY

TUBERCULOSIS IN INDUSTRY

As mills and factories hum and as thousands of men and women swell the ranks of labor under the pressure of war industry, problems concerning the health of workers are in danger of being pushed aside. Tuberculosis is one of the notorious wasters of manpower. Special attention must be given to this disease, which affects and is affected by occupation. To help clarify concepts, a symposium on tuberculosis in industry held at the Saranac Laboratory, Saranac Lake, N. Y., in June, 1941: a résumé. *J. A. M. A.* 8:642–644, 1942. *Tuberculosis in Industry.* 374 pp. New York: National Tuberculosis Association, 1942) was held at the Saranac Laboratory, where leaders in health and industry discussed problems that will also interest the general practitioner. A résumé of the symposium follows.

The prevalence of tuberculosis in any community is determined by the general standard of living and by the number of open carriers. In particular occupations, the factors of selective employment and unfavorable environment modify the picture. If such factors, work involving silica, for example, are dominant, the incidence in the wage earners will be different from that of their families.

The source of the great bulk of infections is a human carrier with a pulmonary cavity. Although the home is probably the place of most childhood and some adult contacts, many primary infections and more reinfections must occur in the place of work. Nurses, physicians and attendants on the sick encounter a real occupational hazard from infection itself; this hazard should be accepted as incidental to the professional life, and hospital management should assume the obligation of minimizing opportunities for mass infection.

About 65 per cent more young women than men die of tuberculosis between the ages of fifteen and twenty-five. From a practical standpoint the employer of large numbers of women needs an effective medical department if he would avoid a tuberculosis problem. Race is a factor to be considered, but it is so intricately associated with the effects of living standards and environment that its effects cannot be weighed. Nutrition is another important factor but also one of the most difficult to evaluate. The influence of fatigue has been studied in the automobile industry and in a steel mill, in neither of which there was evidence that this factor was responsible for any excess of tuberculosis. The belief that abnormal degrees of temperature and humidity lower resistance has little support. Trauma does not initiate a primary infection of the lungs.

Tuberculosis has been regarded as the great enemy of the printer (printers and painters have about 16 per cent more tuberculosis than all other occupied men), and in turn was attributed to lead poisoning, which printers might have contracted. Certain studies indicate that neither lead absorption nor lead intoxication is the cause of excessive tuberculosis among lead and zinc workers.

Fumes and gases are inhalable, and many of them are sufficiently irritating to provoke severe inflammatory reactions. Mature judgment on the effects of gas used by the armies during the last war reversed the early opinion that this agent was responsible for the many cases of tuberculosis that developed. Routine annual examination of a large group of employees engaged in the manufacture of chlorine, phosgene, hydrofluoric acid and other irritating gases supports the view that exposure to irritant gases is not responsible for excessive tuberculosis.

The general thesis that inflammation of the lungs is necessarily unfavorable to the course of associated tuberculosis has little support. It is probably true that certain kinds of inflammatory reactions may have some influence. The increased incidence of tuberculosis that followed epidemic influenza may have been due in part to pneumonic complications.

In grain handlers exposed to high concentrations of organic dust in unloading lake steamers, 2.5 per cent of a group of 234 showed x-ray evidence of clinically significant tuberculosis, and another 2.3 per cent had old healed lesions. Socioeconomic factors, rather than grain dust, were thought to be responsible. Tobacco dust has been under suspicion as a cause of tuberculosis since Ramazzini's studies in 1700. Yet, in a modern cigar factory with a well-organized medical service and air-conditioned rooms, there was less tuberculosis than in the city where the plant was located. Metropolitan mortality figures for 1937–39 show an index for tuberculosis of 107 in cigar and tobacco factory operatives, but it should be noted that 75 per cent of the labor, which now produces only 25 per cent of the product, still works in small shops without health supervision.

Low rates for tuberculosis were found in the Saranac Laboratory studies of the cement and gypsum industries. The usual number of healed lesions was disclosed, so that opportunities for infections had not been lacking.

All these observations support the view that exposure to organic and nonsiliceous dusts has little influence on susceptibility to tuberculosis. Reports on foundries, quartz mining and the granite industry brought out that higher tuberculosis rates prevail in these trades, that there is a greater tendency for such infection to develop after the age of forty rather than earlier, and that the infection is extremely chronic, often giving no symptoms of intoxication or a positive sputum until shortly before death. In miners, the incidence becomes higher and the prognosis of associated tuberculosis worse as the silicotic reaction increases. Miners exposed to silica dust with no roentgenographic evidence of reaction showed little more tuberculosis than the other members of the community in which they lived. Foundries seem to be responsible for the least amount of tuberculosis, whereas the granite industry probably causes the most.

Vermont marble workers had two and a half times as much tuberculosis as the general population of the state (largely rural) exclusive of the granite center in Barre. By contrast, the rate for granite workers was one hundred and thirty times the general one.

The value of a good industrial hygiene program was brought out by the experience of the Eastman Kodak plant. This program costs $10,500 annually, but it also costs $3218 to treat one minimal case of tuberculosis. The attack rate in this plant has fallen from 2.3 at the outset of a study to 0.2 at the present time.

The complexities of compensation insurance carriers were discussed. One plan proposed was that evidence of tuberculosis in any form should preclude employment in industries with silica or other proved hazards and that compensation should be allowed for all tuberculosis subsequently developing in such employment. In other industries, with no specific hazards, persons with healed tuberculosis should be permitted to work, but no compensation should be allowed for infections that might become active or develop during employment. In view of the evidence that old tuberculosis so rarely breaks down in any industry except industries with silica hazards, this appears most equitable.

In the summary, it was pointed out that, aside from nutrition and socioeconomic factors, silica is the only one that has a recognized effect on susceptibility to tuberculosis. Many industrial conditions popularly accepted as predisposing to this disease are without measurable effect. — Reprinted from *Tuberculosis Abstracts*, July, 1942.

NOTE

At a recent meeting of the College of Physicians of Philadelphia, the Alvarenga Prize was awarded to Dr. Edwin J. Cohn, professor of physiological chemistry at the Harvard Medical School, in recognition of his distinguished contributions to the knowledge of blood proteins.

CORRESPONDENCE

COARCTATION OF AORTA

To the Editor: In the December 27, 1934, issue of the *Journal*, I reported two cases of coarctation of the aorta, in father and son. In the former, the clinical diagnosis has been verified.

A. D. did heavy manual labor from the beginning of 1933 until he died on August 16, 1941. He took 1½ gr. of digitalis daily most of the time from 1934 until his death. Extrasystoles were present at each examination. These persisted when he omitted the digitalis. On April 17, 1936, his blood pressure was 192/102; on August 3, 1940, it was 210/106; and on May 10, 1941, it was 176/96. On the last date, he suffered from loss of memory, and moist rales were heard at the bases of the lungs; no paralyses and no abnormal reflexes were found. He was advised to rest and to continue taking digitalis. However, on May 18, he was found shingling a roof. At that time, the blood pressure was 182/94, and the lung bases were clear. In the late afternoon of August 16, the patient's car, which he was driving, collided with another automobile. He turned to his wife and asked how she felt. On being reassured that she was all right, he stepped from the car to the pavement and at once fell down. A physician who arrived shortly thereafter pronounced him dead.

At the request of an insurance company, the patient's body was exhumed, and an autopsy was performed by Dr. Benjamin Castleman on October 9. Just below the attachment of the closed ductus arteriosus, the lumen of the aorta was completely blocked. There were hypertrophy of the left ventricle and marked sclerosis of the coronary arteries. The aortic cusps were normal. The internal mammary arteries were four times the usual size. The abdominal aorta and the iliac arteries were small.

Immediately after the accident, and at the post-mortem examination, there was no evidence of severe traumatic injury.

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